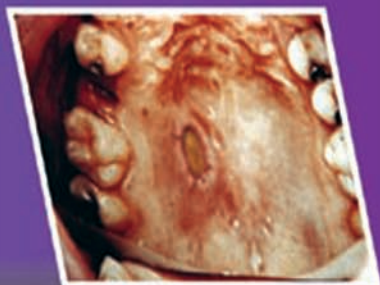




Oral Diseases and Disorders

Differential Diagnosis

SR Prabhu



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***Oral Diseases
and Disorders***

Differential Diagnosis

Oral Diseases and Disorders

Differential Diagnosis

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Oral Diseases and Disorders: Differential Diagnosis

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Preface

Diagnosis of diseases and disorders involving tissues and organs of the body is often inconclusive without the differential diagnosis. Differential diagnosis based on clinical, radiological, histological and other investigative routes forms an important part in any branch of medicine. Students of dentistry need to learn relevant aspects of diagnostic process during their training.

Oral Diseases and Disorders: Differential Diagnosis is an attempt to provide relevant information on differential diagnosis of various conditions that involve oral and maxillofacial structures. Style, design and structure of chapter presentation have been kept simple and controversial aspects that relate to some oral diseases have been deliberately omitted. Relevant color and black and white illustrations have been used to complement the text.

This book is meant to serve primarily the undergraduate students of dentistry. Students preparing for Oral Diagnosis, Oral Medicine and Oral & Maxillofacial Surgery Examinations will find this book useful. It is hoped that practitioners of dentistry and those pursuing dental hygiene courses also will find this book useful.

July 2007

SR Prabhu

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CHAPTER

1

Essentials of the Diagnostic Process

SR Prabhu

Learning objectives

After studying this chapter the student should be able to:

- Understand the meaning of the word diagnosis
- Explain different types of diagnoses that are in use
- Describe the basic steps involved in the process of diagnosis.

Introduction

The process of establishing the nature of an abnormality or disease that produces signs and symptoms is called Diagnosis. Modern therapy rests on accurate diagnosis. Students of dentistry therefore must learn the importance of systematic approach and methods involved in collecting the necessary material that enables them to make correct diagnosis.

To qualify its meaning, often adjectives are added with the word diagnosis. Examples include:

- *Clinical diagnosis:* This implies that the diagnosis is made on the basis of information gathered through history and physical examination.
- *Radiographic diagnosis:* Diagnosis in this case is made on the basis of radiographic findings.
- *Microscopic diagnosis:* This implies that the diagnosis is made on the basis of microscopic features.
- *Working or tentative diagnosis:* This means that the available information points to a certain diagnosis but further diagnostic studies will be necessary to substantiate this impression.
- *Differential diagnosis:* This involves a list of diseases or conditions in order of diagnostic probability. The diseases listed share certain common features. The list begins with the most likely of all the relevant diseases or conditions that

should be considered. In formulating a differential diagnosis, it is important to mention evidence that is for and against each of the entities that are listed.

- *Final diagnosis:* This is the definitive diagnosis made on the basis of all available observations and laboratory investigations.

Once the diagnosis is established, the clinician will know what further steps to take. The information gathered also provides the clinician with the knowledge of a forecast of the probable course or the outcome of the disease with or without treatment.

Most important factors which determine student's/clinician's ability to make a diagnosis include:

- The knowledge of how to conduct a thorough search of a problem
- The knowledge of how to observe and differentiate deviations from normal
- The knowledge of diseases or conditions that might be causing the problem.

Basic approach to diagnosis

It must be remembered that a systematic and scientific approach to the patient's problems is central to the diagnostic process. There are four major steps to the diagnostic process. They are:

1. Collection of data from the patient's history, physical examination and from relevant adjunctive diagnostic procedures.
2. Analysis of information gathered to assess its clinical implication.
3. Formulation of a differential diagnosis.

4. Making a final diagnosis on the basis of information gathered from the history, physical examination, differential diagnosis and diagnostic tests.

Collection of information

History

History is obtained from the patient by questioning. It is generally accepted that 85 to 90 percent of diseases can be discovered by a well-conducted health history. Patient is encouraged to provide a narrative of his/her health problems in chronological order from the onset to the present time. Patients' problems are called symptoms. Symptoms are subjective manifestations of disease and represent problems such as pain, swelling, and difficulty in opening the mouth and so on. History taking is a valuable information gathering process.

Physical examination

Physical examination involves different clinical methods employed to identify changes produced by the disease. Structural and functional changes brought about by the disease can be observed (inspection), felt (palpation) and tested by other clinical examination methods. These changes can also be identified by the use of relevant diagnostic aids. Changes detected by the clinician using any of these methods are referred to as signs. Signs of the disease therefore are objective manifestations of disease. Ulceration, swelling, blisters are some of the examples of signs. In this context, an appreciation of the normal is necessary. Clinician must also

possess adequate knowledge and skills of identifying abnormal structures from the normal and from the structural variations. For example, Fordyce's spots (ectopic sebaceous glands) lying superficially on the posterior part of the buccal mucosa are often mistaken for disease. Presence of these structures is not a sign of disease.

Adjunctive diagnostic tests

Diagnostic tests offer supportive evidence to reach a final diagnosis. It must be remembered that the tests are not to be ordered on a routine basis. X-rays and clinical laboratory tests, including blood tests, microbiologic tests and urinalysis are some examples in this category. Microscopic examinations carried out on biopsy specimens are of immense value particularly in providing confirmatory evidence of life threatening conditions such as oral cancer. It must be borne in mind that the diagnostic tests should not replace history taking and physical parts of the patient evaluation process.

Analysis of information

Data that have been collected from the history, clinical examination and diagnostic laboratory tests need to be evaluated in an organized manner in order to be able to arrive at a diagnosis. Process of data evaluation is an ongoing procedure and starts quite early in the diagnostic process. Every bit of information gathered is significant even if the finding is negative. Analysis of information provides useful clues to understand the nature of the disease process as to whether these are inflammatory, degenerative,

or neoplastic and so on. This categorization of disease entities limits the number of diseases to be considered in differential diagnosis.

Formulating differential diagnosis

Differential diagnosis essentially involves consideration of a host of diseases or conditions that share similar features and eliminating one by one based on the evidence available. Differential diagnosis leads to a working diagnosis. In general, a list of differential diagnosis is established once the database from history and physical examination has been completed. In some conditions however, signs and symptoms may indicate a primary bone disease involving the jaw or point to a systemic process. In these situations radiographs, biopsy or other relevant laboratory diagnostic tests may be required.

Making a final diagnosis

Collected facts need to be sorted out in order to arrive at a diagnosis. This process requires a deductive reasoning on the part of the clinician. As mentioned above, this process of diagnosis is approached through a process of elimination on the grounds of changes produced by the disease. The diagnosis thus made may be provisional and to arrive at a definitive diagnosis, further investigations may be required.

Once a final diagnosis has been made, the most appropriate treatment is selected. Depending on the diagnosis made, a number of management options are available to the clinician, in general they include:

- No treatment
- Palliative treatment

- Curative treatment
 - Therapeutic
 - Surgical
- Functional treatment
- Preventive treatment and
- Follow-up.

Suggested reading

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CHAPTER

2

Diagnostic Sequence

SR Prabhu

Learning objectives

After studying this chapter the student should be able to:

- Understand the reasons why patients seek oral care
- Know the order in which sequential clinical examination should be undertaken by the diagnostician
- Know the components of general appraisal of the patient
- Know what constitutes the examination of extraoral structures
- Know what constitutes intraoral examination
- Know how to conduct examination of the symptomatic patient
- Know how to categorize the oral diseases in the diagnostic process.

Introduction

Early in their clinical training students are uncertain as to where to start and how to proceed with the clinical procedures. This uncertainty is understandable. By the passage of time, however, through study and practice, students gain confidence and the clinical procedures they perform on patients become smoother and systematic.

It is important that during their early part of clinical training, students should repeatedly perform a comprehensive and sequential examination in order to gain proficiency in all aspects of examination methods. Clinicians should also perform detailed clinical examination of their patients periodically so as to maintain their clinical skills.

A systematic, sequential and comprehensive examination has two major purposes:

1. It serves the patient by minimizing his/her discomfort and
2. It maximizes the examiners' efficiency.

Variations and ‘shortcuts’ in the order of examination are acceptable only when the presenting symptoms demand urgent attention by the clinician. For a patient who has acute symptoms restricted to a tooth for example, an extensive examination that includes structures of the head and neck would be unnecessary at that visit. Indeed, the examiner should select the relevant method to assess the problem as precisely as possible and treat the condition at the earliest.

Sequencing the examination procedures is an important aspect of oral diagnosis. Examination sequence should be systematic, scientific and logical. Haphazard examination protocols have no place in any clinical discipline. An orderly, systematic approach makes the examiner less susceptible to errors of omission.

Patient category

Patients seeking oral care broadly fall under the following categories:

- Those seeking diagnosis and treatment for specific complaint(s) such as tooth decay, bleeding gums, pain, swelling, ulcer, etc
- Those seeking a routine dental check-up
- Those seeking periodic oral hygiene maintenance or preventive care
- Those seeking appliances and prosthesis, etc.

Although most dental patients regard detailed clinical examination with some degree of anxiety, they often appreciate clinicians’ full attention of their problems.

As a prelude to the chapters that will follow in the first section of the book, this chapter is intended to

provide an outline and an overview of the comprehensive and sequential physical examination involved in oral diagnosis.

Having carried out the history and patient interviewing part of the diagnostic process, the comprehensive clinical examination should be carried out in a sequential manner. An outline is shown below:

General appraisal of the patient

- General survey of the physical and mental status
- Determination of the vital signs
- Gross examination of the skin, hands and nails.

Examination of the specific extraoral structures

- Examination of the head
- Examination of the face
- Examination of the neck
- Evaluation of the cranial nerve function.

Examination of the intraoral structures

- Assessment of the overall oral hygiene status
- Examination of the intraoral soft tissues and evaluation of the salivary function and taste sensation
- Examination of the teeth, dentition and occlusion
- Examination of the periodontium.

Brief account of the above procedures is given in the following paragraphs:

General appraisal of the patient

Evaluation of the patient's general state of health is

important for several reasons. Important aspects among these are:

- Protection of the patient from harm that might be caused by dental treatment
- Protection of the dentist and his/her staff from possible contagious diseases such as hepatitis B
- Opportunity for the examiner to suspect or detect early systemic disease that the patient is unaware of.

General appraisal of the patient is carried out by obtaining information on the following:

1. Stature
2. Body type
3. Posture
4. Gait
5. Mobility
6. Symmetry
7. Sexual development
8. Color
9. Skin characteristics
10. Facial expression
11. Hair
12. Hands and nails
13. Verbal and non-verbal responses
14. Mental status
15. Determination of the vital signs: blood pressure, pulse; body temperature and respiratory rate.

Normally, these procedures do not take more than fifteen minutes of the examiner's time.

Outline of the comprehensive examination and its sequence

Examination of the specific extraoral structures

Structures to be examined include:

- *Head:*
 - Hair
 - Scalp
 - Skull
- *Face:*
 - Facial form and skin
 - Pre-auricular and post-auricular lymph nodes
 - Eyes
 - Ears
 - Nose
 - Sinuses
 - Temporomandibular joint
 - Maxilla and
 - Mandible
- *Neck:*
 - Cervical lymph nodes
 - Thyroid cartilage
 - Cricoid cartilage
 - Hyoid bone
 - Trachea
 - Thyroid isthmus and the gland
 - Neck muscles
 - Carotid pulsation

Evaluation of the cranial nerve functions

This examination process aims at assessing the sensory and motor functions of the cranial nerves. Evaluation of the following functions is carried out during the clinical examination:

- Strength of the muscles of mastication and facial expression

- Corneal reflexes
- Sense of smell.

Examination of the intraoral structures

Intraoral clinical examination should be carried out in the following orders:

Soft tissues

- Vermilion border
- Labial mucosa
- Buccal mucosa
- Vestibular mucosa
- Alveolar mucosa
- Gingiva
- Palate
- Soft palate and uvula
- Oropharynx
- Tongue: Dorsal, lateral
- Floor of the mouth and ventral surfaces of the tongue

Teeth dentition and occlusion

- This includes a thorough clinical examination of individual teeth for their morphological and structural characteristics
- Dentition for occlusion and
- Tooth vitality where appropriate.

Periodontium

- Gingival tissues
- Gingival sulcus
- Tooth mobility to determine the status of periodontal health.

Examination of the symptomatic dental patients

When a lesion or discomfort such as pain or ulcer is detected, the physical examination needs to be appropriately modified. Important steps that are to be followed include:

- Analysis of chief complaint
- Classification of the disease process
- List of possible diagnoses
- Development of the differential diagnosis
- Development of the clinical impression
- Formulating a final diagnosis.

Analysis of chief complaint

Symptom analysis is an essential step before commencing a detailed clinical examination of the presenting lesion or discomfort. Symptomatic patients seeking oral care may complain of one or more of the following problems: dental pain, swelling, ulcers, blisters, loose teeth, temporomandibular joint problems, occlusal problems, dry mouth, burning mouth, oral malodor and other less common symptoms.

Prior to starting the examination process examiner gathers information on the symptoms with regard to their location, duration, nature, severity and other characteristics.

At this point it is important to view the diagnostic process as a method of problem solving.

Once the examiner has recognized the presence of an abnormality he/she proceeds to examine it using several clinical methods. These essentially include:

- Inspection

- Palpation
- Percussion
- Auscultation and probing.

Classification of the disease process

The manifestations and types of diseases encountered in patients are many and diverse. An individual dental practitioner may not personally see many of the rare diseases described in various textbooks. This does not mean however, that a student of dentistry does not have the responsibility to possess knowledge and understanding of the broad range of oral diseases.

One way of solving problem in a clinical setting is to devise systems or classifications of diseases. Indeed, there is no single classification that provides an easy way of learning the relevant clinical and pathologic features of the whole range of diseases. Also, it is true that neither does any one system or classification suit all clinical students or practitioners. However, there are classifications that have stood the test of time, which provide a reasonably good means of learning the disease process.

Having gathered information through the process of history taking patient interview and clinical examination, the examiner then puts the facts into various categories.

One can label this categorization method—a surgical sieve. The surgical sieve is a useful diagnostic aid. Basically, it is recognition that all of the different diseases affecting patients can be categorized into a few groups according to the nature of the underlying pathology. Surgical sieve is a system, which can be

applied, to “disease” as a whole or to disease of an organ or tissue (such as that of jaw bone, salivary gland, oral mucosa and so on). When a practitioner is confronted with a patient who has a lesion or a condition requiring diagnosis, the surgical sieve can act as a mental checklist.

The surgical sieve also helps to categorize all primary diseases of the organ or tissue into one of the following groups:

- Developmental
- *Inflammatory*:
 - Infectious
 - Immunologic
- Hyperplastic
- Degenerative
- Hormonal/Metabolic
- Neoplastic or
- Idiopathic.

Above listed major categories do have limitations. For example, degenerative and neoplastic categories indicate the general nature of the pathology and do not shed light on the etiology. Hormonal or metabolic group tells us about etiology and nothing about pathology. It is also true that a number of acquired hyperplastic lesions (of the oral mucosa for example) have an inflammatory component. It is important that a student must recognize such limitations.

When used as a clinical aid to diagnosis an additional category should be added to the surgical sieve namely: “Oral Manifestations of Systemic Disease”. Indeed, used in this way any disease or lesion included under this particular group can itself be grouped under one or more of the groups listed above

(such as developmental, inflammatory, hyperplastic and so on).

Another method employed in diagnosis is to categorize lesions according to their predominant clinical feature or presentation. In this way categories can be derived as follows:

- White lesions
- Ulcers/Erosions
- Vesicles/Bullae
- *Swellings*:
 - Localized
 - Diffuse
- Pigmented lesions
- Clefts
- Changes in shape and size of oral structures and so on.

Bony lesions when encountered, they can be grouped on their radiographic appearances such as radiolucent, radiopaque or mixed (radiolucent and radiopaque combined) lesions.

List of possible diagnoses and developing differential diagnosis

Categorization of the diseases as discussed is highly useful at the clinical level. A basis for differential diagnosis can be developed through this method of categorization.

Indeed, to use this system effectively, examiner should be aware of the broad range of pathology, which can affect the organ or tissue. Once this is achieved, it is then simply a matter of making a “list” of lesions or diseases, which can be grouped together under headings such as those, listed above.

For example, under white lesions the list of lesions that can be prepared includes frictional keratosis, idiopathic leucoplakia, lichenplanus, candidiasis, chemical burn and so on. Used properly, this system allows the student to 'rattle off' when asked, a list of names of lesions or diseases, which present clinically as 'white lesions'.

Indeed, as a prerequisite the examiner should be aware of the full range of pathologies and their clinical similarities and differences. Developing differential diagnosis will not be effective otherwise.

Developing a clinical impression and formulating a final diagnosis

Systems such as the surgical sieve and preparation of lists of lesions which share common clinical/radiographic expression do not of themselves eliminate the need to 'learn' the various clinical and pathologic features of individual diseases and lesions. It is of little benefit if a student is able to rapidly think of a list, (for example, the names of conditions which present as 'white lesions' of the mucosa) but does not know the various features of the different conditions and how he/she can distinguish one lesion from the other using a clinical or other pathologic criteria. Student is required therefore to know the following with respect to specific lesions.

1. *Clinical features of the lesions that include:*
 - Relative frequency of occurrence, race, age and gender distribution where applicable
 - Site distribution

- Symptoms and signs. For example:
If it is an ulcer: is it painful/tender? Single/multiple? Is it recurrent? What are its characteristics in terms of: size, shape, location, edge, border, floor, color, surrounding tissue and so on?
- 2. *Pathology of the lesion that includes:*
 - Nature (e.g. developmental, inflammatory etc.)
 - *Etiology:* Specific, predisposing factors if applicable
 - Pathogenesis—What are the biologic processes occurring?

Final diagnosis

Having developed a working diagnosis on clinical grounds it is important to further examine the lesion to recheck the credibility of the clinical impression. If necessary patient should be asked more questions. At this point the examiner decides on ordering additional tests (radiographs for bony lesions for example) if required.

In many cases of oral disease, microscopic diagnosis provides confirmation of the working clinical diagnosis. Indeed, other clinical laboratory tests may also contribute in offering clues to a final diagnosis in certain situations. A student therefore should also possess adequate knowledge of all the relevant aspects of investigative pathology.

Suggested reading

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CHAPTER

3

**Key Features
in Establishing
Source of
Orofacial Pain**

SR Prabhu

Learning objectives

After studying this chapter the student should be able to:

- Understand the nature of orofacial pain of oral structures
- Know how to differentiate pain of dental and non-dental sources.

Introduction

The most common reasons why many persons seek dental consultation include pain of dental and oral structures. Patients with orofacial pain present a real diagnostic and therapeutic challenge to the practitioner because of the complex nature of pain.

Odontogenic source of pain is more frequent. Clinician often finds it difficult to differentiate between the odontogenic and non-odontogenic nature or source of orofacial pain.

Pain is a subjective symptom. A systematic approach to history and signs and symptoms of the presenting pain is of great importance in providing a clinical diagnosis. The features can be distinct to each category or may be common to both categories.

The differentiation between odontogenic and non-odontogenic must be established first.

If the pain is found to be of dental origin, look for dental pathology and differentiate between pain of pulpal and periodontal origin.

Evaluation of orofacial pain

Evaluation of orofacial pain begins with:

- Comprehensive history taking
- Physical examination with special reference to

orofacial structures and or detailed neurological examination

- Behavioral and psychological assessment of the patient
- Diagnostic tests.

Briefly some aspects of evaluation of orofacial pain are discussed here.

A comprehensive history format for orofacial pain patients includes the following:

- *Chief complaint—describing pain*
 - Location (localized, diffuse, does it move)
 - Intensity (severe, mild)
 - Nature (stabbing, burning, dull, throbbing, etc.)
 - Duration (when did it start?)
 - Influences of external factors such as time of the day, jaw motion, body position, cold, etc.
 - Modifying factors (alleviating, precipitating or aggravating)
 - Remissions or change over a period of time
 - Previous treatment results.
- *Medical history and review of systems*
 - Current or pre-existing disorders such as arthritides or musculoskeletal disorders; diabetes, etc.
 - Previous surgeries/treatments
 - Trauma to the head and face
 - Medications
 - Allergies/skin disorders
 - Alcohol and other drug abuse
 - Psychiatric problems
 - Maxillofacial or ENT problems.

- *Dental history*
 - Current or pre-existing relevant dental disorders
 - Previous dental treatments
 - Habit history such as bruxism, clenching, excessive gum chewing, nail biting, etc.
- *Psychosocial history*
 - Social, behavioral and psychologic, occupational and family, relationships, etc.

Comprehensive physical examination

- General inspection and palpation of the head and neck region
 - Symmetry, size, shape and movement of the head, neck and face
 - Conducting vascular compression of the temporal and carotid arteries.
- Evaluation of temporomandibular joint and associated musculature
 - Palpation of muscles of mastication
 - Palpation of TMJ externally and through external auditory canals (with mouth closed and opened)
 - Measurement of the range of motion, quality of movement and association with pain
 - Auscultation for TMJ noises
 - Identification of any tenderness, swelling unusual texture, etc.
- Neurologic evaluation
 - Conducting cranial nerve screening
- Ear, nose and throat evaluation
 - Inspection of ear and nose for pathology
 - Examination of oropharynx.

- Intraoral evaluation
 - Hard tissue examination
 - Soft tissue examination
 - Analysis of occlusion—both static and dynamic.
- Comprehensive diagnostic tests.

Radiography

- Intraoral views to rule out dental and periodontal pathology
- Extraoral views to view TMJ and other osseous structures.

Clinical/laboratory tests (less common)

- Blood testing for rheumatologic pain and those of infectious origin
- Arthrocentesis (aspiration of synovial fluid)
- Diagnostic anesthetic injections
- Biopsy for pain originating from soft tissue lesion or intraarticular biopsy for TM joint lesions.

Character of pain and its association with orofacial conditions

Sharp, stabbing pain

Sharp, stabbing pain which is generally of short duration is a feature conditions such as:

- Fractured tooth or restoration
- Hypersensitivity of dentine
- Exposed dental pulp (acute pulpitis) due to caries
- Non dental causes such as trigeminal and glossopharyngeal neuralgias. Sometimes triggering zones are associated with pain. Examples include swallowing act in glosso-

pharyngeal neuralgia and face shaving or splashing water on the face for trigeminal neuralgia

- Thermal changes and sensitivity to sweet, sour are known to cause pulpal pain in a carious tooth or where dentine is exposed to the oral environment
- Sight of food or eating causes salivary gland pain in salivary gland duct obstruction due to stones
- Intermittent pain due to release of pressure from biting is a feature of cracked tooth syndrome.

Dull, throbbing pain

Dull throbbing pain may last for hours or days. This is a feature of:

- Chronic pulpitis
- Periodontal inflammation
- Pericoronal infections
- Necrotizing ulcerative gingivitis/stomatitis
- Dry socket, sinusitis
- Atypical odontalgia
- Atypical facial pain
- Herpetic infections
- Cardiogenic pain
- Temporomandibular disorders
- Neoplasms, and
- Various types of oral ulcers.

Burning pain

Mucosal burning is felt in burning mouth syndrome and as prodromal symptoms of shingles and recurrent herpes virus infections. Burning is also

experienced in allergic stomatitis and radiation mucositis.

Pain scale

Patients should be asked to express severity of pain on a scale of 0-10. Zero represents no pain while number 10 on the scale represents pain of worst intensity.

Some key points to remember in evaluating orofacial pain:

- Pain arising from dental causes and periodic episodes of migraine affect sleep
- Severity of pain in these instances may increase on the body position such as lying flat or bending forwards
- Atypical facial pain and trigeminal neuralgias on the other hand do not affect sleep
- Unilateral pain is generally of dental origin while bilateral pain may be a feature of nondental causes such as sinusitis, burning mouth syndrome and atypical facial pain
- Periodontal pain is localized whereas pain due to pulpal pathology is not localized. In the case of periodontal pain the patient is able to point out the offending tooth whereas in painful condition due to pulpal pathology the patient is not able to point out the offending tooth; the pain may even be referred to other teeth on the same side. Injection of diagnostic local anaesthetic agent is helpful in locating the pain in such instances.
- Response to analgesics in the relief of sharp pain is generally poor, dull pain responds better to the use of analgesics

- When infection is associated with pain other cardinal signs of inflammation such as swelling, raised local temperature and loss of function may be present. In addition, regional lymph node enlargement, pus discharge from the swollen parts and fever may also present in these cases.

CHAPTER

4

Differential Diagnosis of Pain of Dental Origin

SR Prabhu

This chapter is reproduced in part from: SR Prabhu, Mysara Shawaf: Orofacial Pain: SR Prabhu (Ed) 2004: In Textbook of Oral Medicine: Oxford University Press 48-59.

Learning objectives

After studying this chapter the student should be able to:

- Know the nature, causes and management of orofacial pain of dental origin
- Differentiate between pain of dentinal, pulpal and periodontally derived pain.

Classification of pain of dental origin

The most common pain of dental origin is pulp pain or pulpalgia. This may be classified according to the degree of severity as well as the pathologic process present.

The most practical classification is as follows:

1. *Pain of mild pulpal stimulation:* Hyperreactive pulpalgia
 - Dentinal hypersensitivity
 - Hyperemia.
2. *Pain of acute pulpal inflammation:* Acute pulpalgia; acute pulpitis:
 - Incipient
 - Moderate
 - Advanced.
3. *Pain of chronic pulpal inflammation:* Chronic pulpalgia; chronic pulpitis:
4. Pain of hyperplastic pulpitis.
5. Pain of necrotic pulp.
6. Pain of internal tooth resorption.
7. Pain of traumatic occlusion.
8. *Pain of incomplete tooth fracture:* split tooth; cracked tooth syndrome.
9. *Pain of periapical pathology:* Periradicular pain
 - Acute apical periodontitis
 - Acute apical abscess

- Chronic apical periodontitis
 - Chronic apical abscess
 - Apical cysts.
10. Postoperative dental pain.
 11. Referred pain.

Hyperreactive pulpalgia

- Hyperreactive pulpalgia is characterized by a short, sudden sharp “shock” usually elicited by an exciting factor
- The pain is of short duration lasting as long as the exciting factor is in contact with the tooth
- The sensations of hyperreactive pulpalgia may be divided into two types namely: hypersensitivity and hyperemia.

Hypersensitivity

- Caused by dentine exposure
- Exciting factors of a hypersensitive pulp are usually many. These include: cold food drink, cold air, contact of two dissimilar metals or stimulation of the exposed dentin on the root surface by cold, sweet or sour substances, fruit acid, salt, glycerine or the touch of the surface with a finger nail or tooth brush or an explorer
- In many instances, patient is able to point out to the sensitive tooth.

Hyperemia

Hyperemia involves increased blood supply in the pulp.

- When heat is applied to the pulp hyperemia results eliciting dull pain. This does not happen when cold is applied

Variations in dentin sensitivity

In clinical practice some variations in dentin sensitivity are expected under the following conditions:

- Molars are usually less sensitive than canines or pre molars
- Posterior teeth are usually less sensitive than incisors
- Older teeth are less sensitive than younger teeth.

Dental plaque removal should be the first step before any desensitizing treatment is undertaken.

Treatment

Treatment of hyperreactivity involves chemical/mechanical obstruction of dentinal tubules. Agents which give satisfactory results are: Potassium oxalate, (sold commercially as protect), strontium chloride, (contained in “sensodyne: tooth paste) sodium and stannous fluoride and resins including the new adhesives. Fluoride iontophoresis is successful for dentinal hypersensitivity.

Acute pulpal pain (acute pulpalgia)

Acute pulpal inflammation results in acute pulpitis producing intense pain. Degree of severity in acute pulpalgia can be grouped into three categories: incipient, moderate and advanced.

Incipient acute pulpalgia

- Mild discomfort experienced as the anesthetic wears off following cavity preparation can be regarded as an example of incipient acute

pulpalgia. This sensation disappears in a matter of few hours

- Traumatic occlusion, cold and sugar are the other known agents which stimulate incipient acute pulpalgia.

Moderate acute pulpalgia

- This is a true toothache but the one that patient can tolerate
- The pain is “boring” in nature and is localized to start with
- The pain usually does not disappear soon after the irritant is removed. It may persist for a few minutes or hours
- The pain may start on change of posture—for example, while lying down or bending over
- It is likely that any act that increases cephalic blood pressure will start the pain
- Often eating something cold starts moderate acute pulpalgia
- A warm water rinse does not usually relieve the pain, cold water makes it worse
- Analgesics such as aspirin relieve pain temporarily
- Pain of moderate acute pulpalgia is difficult to pin point after a few days of discomfort
- Clinical examination involves pulp testing and radiography
- Percussion and palpation are not very useful
- Cold testing followed by heat testing is to be carried out. The immediate response to cold is quite sudden, violent and lasting, while heat application is not conclusive in eliciting pain.

Treatment of moderate acute pulpalgia includes pulpectomy and endodontic therapy. The use of potassium nitrate as a desensitizing agent is often helpful.

Advanced acute pulpalgia

In advanced acute pulpalgia patient experiences one of the most excruciating acute pains in the tooth.

- The relief of advanced acute pulpalgia can be achieved by cold water rinse. This is indeed a temporary measure. The relief may last only for 30-40 seconds. Patient may have to continue to rinse cold water until he/she seeks dental care. If cold aggravates pain, the patient has moderate acute pulpalgia.

Other features include:

- Offending tooth can be pointed out by the patient
- Affected tooth is mildly tender on percussion
- Tooth is less sensitive to the pulp tester requiring higher reading
- Pain aggravates with heat application (hot gutta-percha)
- Local anesthesia relieves the pain.

Treatment of advanced acute pulpalgia includes pulpectomy followed by endodontic therapy. Extraction is indicated if the tooth cannot be saved.

Chronic pulpalgia (chronic pulpitis)

In chronic pulpalgia, patient can withstand the pain for weeks or months.

Key features are:

- Pain is diffuse

- Chronic pulpalgia may cause referred pain
- Chronic pulpalgia may result in an abscess
- Pain may aggravate on changing the position such as bending over or lying down.
- In chronic pulpalgia there may be a large carious lesion present, there may be a fractured amalgam restoration or recurrent caries under a restoration
- Recurrent caries under a full crown or a three quarter crown can give rise to chronic pulpalgia. This is difficult to test using thermal or electrical tests or by radiographs. CO₂ “ice” is useful in these circumstances
- Pulp tester and radiographs are the best diagnostic tools for chronic pulpalgia. Maximum discharge from the electric tester is needed in detecting chronic pulpalgia
- Radiograph reveals interproximal or root caries or recurrent caries under a restoration
- Chronic pulpalgia often exhibits referred pain in the region
- Treatment of chronic pulpalgia is pulp extirpation and endodontic therapy. If the tooth cannot be saved, tooth should be extracted.

Hyperplastic pulpitis

- Hyperplastic pulpitis or pulp polyp is usually painless.
- The hyperplastic tissue can be lifted away from the tooth walls with the help of a spoon excavator.
- Majority of cases exhibit badly decayed unrestorable tooth.
- Diagnosis of hyperplastic pulpitis is easy.

Necrotic pulp

- Complete necrosis results in symptomless tooth
- When pulp is partially dead (necrotic) the symptoms are those of chronic pulpalgia
- Clinical examination of total necrosis of pulp reveals coronal discoloration
- X-rays help only if periapical lesions are present due to necrotic pulp
- It is always important to carry out pulp testing even if periapical pathology has resulted in a case of pulpal necrosis.

Treatment includes endodontic therapy if the tooth deserves to be saved.

Pain due to internal tooth resorption

In internal tooth resorption, the pulp can be symptom free or elicit pain similar to that of chronic pulpalgia.

Two methods are used to detect internal resorption:

(1) visual examination which may reveal “pink spot” through the enamel or (2) radiography which reveals resorption of the tooth and root form within.

Treatment of internal resorption involves pulpectomy.

Pain due to traumatic occlusion

- Tooth traumatized due to bruxism or because of a restoration that is in hyperocclusion can give rise to mild pain similar to that of chronic pulpalgia
- Patient with bruxism may complain of vague pain in the tooth/teeth upon awakening in the morning.
- Thermal and electric pulp test responses are like that of normal teeth

- Presence of wear facets give further evidence of bruxism
- Teeth may be mildly painful on mastication or chewing on a cotton roll.

Treatment for “high points” of restoration includes relief of occlusal trauma by grinding the involved and/or opposing teeth.

Pain due to incomplete tooth fracture (split tooth, cracked tooth)

The tooth that is split or cracked but not fractured often presents a range of symptoms from hypersensitivity to continuous pulpalgia.

- The pain of cracked tooth may be felt during mastication as one quick unbearable stab
- True pulpitis results if the split has crossed through the pulp
- Contacting cold fluids often elicits pain in these patients
- Diagnosis involves identification of the split and its extent. Heat or cold may not yield much help. Percussion may not yield much clue either. Biting on cotton roll elicits pain and this test is of help. Thermal and electric pulp testing may be normal
- Crack can sometimes be made visible by painting the tooth with tincture of iodine which is washed after 2 minutes. The crack often appears as a dark line
- Radiographs are not of much help.

Treatment involves preparation of a full crown if pulp is not involved. In the case of pulp involvement, pulpectomy, endodontic therapy and preparation of a full crown is required.

Postoperative pain

Often postoperative pain is associated with various treatments carried out in the orofacial region. Endodontic therapy frequently results in post operative pain due to apical overextension of necrotic debris or instruments. Other operative procedures in the orofacial region are also responsible for postoperative pain. History is always suggestive in these cases.

Periradicular pain

Acute apical periodontitis (AAP)

- Pain of acute apical periodontitis can be excruciating and lasts for a few days
- Tooth is very tender to touch. Pain is constant, throbbing or pounding
- The tooth may be in supraocclusion.

Based on the clinical symptoms and signs diagnosis of acute apical periodontitis is relatively easy. Treatment involves correction of occlusion under local anesthesia. Use of systemic anti-inflammatory agents and analgesics also are of great help in relieving symptoms.

Acute apical abscess (AAA)

- The pain of acute apical abscess is less intense as compared to that of acute apical periodontitis
- Throbbing pain particularly on palpation or mastication is felt by the patient.

Often patient may have an observable swelling due to the eroded alveolar plates by the spreading abscess. Occasionally this may give rise to cellulitis.

- The involved tooth is extremely sensitive to percussion
- Radiographs may show a widened periodontal space or an area of a alveolar radiolucency
- Electric pulp testing of the tooth confirms necrotic pulp as being responsible for apical abscess.

Treatment involves establishment of drainage either through the root canal or by incision if the abscess is fluctuant. Systemic antibiotics and analgesics are important. The offending tooth requires either endodontic treatment or extraction depending upon the extent of abscess.

Chronic apical periodontitis (CAP)

- Pain of chronic apical periodontitis is mild
- In most cases, this occurs as a sequela to pulp necrosis
- Radiography may show visible bone resorption. Resorption of lamina dura and thickening of periodontal ligament space are visible on X-rays
- Pulp vitality tests elicit no significant pain.

Endodontic therapy is usually indicated. Sometimes periradicular surgery may be required.

Chronic apical abscess (CAA)

- This is also called suppurative apical periodontitis
- Usually, this condition is symptom-free
- There may be a draining abscess through a sinus tract
- Occasionally, chronic apical abscess may develop into an acute exacerbation called phoenix abscess
- Chronic apical abscess may be associated with old dental restorations or extensive bridge work

- Radiologically chronic apical abscess reveals diffuse radiolucency around the apex of the involved tooth
- External tooth resorption of the root may also be seen in majority of these cases.

Treatment of chronic apical abscess involves endodontic therapy.

Suggested reading

1. Balciunas BA, et al. A clinical approach to the diagnosis of facial pain. *Dental Clinics of North America*:WB Saunders Co, Philadelphia 1993;37:31-71.

CHAPTER

5

Differential Diagnosis of Pain of Non- Dental Origin

SR Prabhu

This chapter has been reproduced in part from: SR Prabhu (Ed): SR Prabhu and Mysara Shawaf. Orofacial Pain. In Textbook of Oral Medicine: Oxford University Press 2004;48-59.

Learning objectives

After studying this chapter the student should be able to:

- List non-dental sources of orofacial pain
- Know in detail how to diagnose different types of neuralgias
- Know how to differentiate between painful conditions originating from maxillary sinus, TMJ, salivary gland and other non-dental sources.

Introduction

Orofacial pain of non-dental origin is often difficult to diagnose. First of all possibility of odontogenic origin of pain should be eliminated prior to establishing a diagnosis of pain of non-dental origin. Reader is therefore referred to chapters 3 and 4 for a detailed discussion on this subject.

Conditions discussed in this chapter include:

- Orofacial neurogenic pain (such as different types of neuralgias)
- Orofacial pain of vascular origin
- Orofacial pain of muscular origin
- Orofacial pain due to tension type headache
- Orofacial pain of temporomandibular joint origin
- Orofacial pain due to growth disorders
- Burning mouth syndrome
- Orofacial pain of salivary gland origin
- Orofacial pain originating in the ear
- Orofacial pain of maxillary sinus origin
- Orofacial pain of cardiac origin.

Maxillofacial pain of non-odontogenic origin based on the anatomic location is shown in the Table 5.1 below:

Table 5.1: Differential diagnosis of non-odontogenic maxillofacial pain according to anatomic location

<i>Anatomic location</i>	<i>Cause of pain</i>
Frontal	Sinusitis, Ocular disorder Muscle tension headache Migraine headache Cluster headache, Post-herpetic neuralgia
Temporal	Migraine headache, Temporal arteritis, TMO, Hypertension headache Post-herpetic neuralgia
Occipital	Muscle tension headache Cervical arthritis, Hypertensive headache
Orbital	Ocular and/or optic disorder Cluster headache, Migraine headache Sinusitis, Post-herpetic neuralgia
Nasal/paranasal	Glossopharyngeal neuralgia Sinusitis, Trigeminal neuralgia
Maxilla	Sinusitis, Odontalgia Trigeminal neuralgia Lesions in maxillary sinus
Mandible	Glossopharyngeal neuralgia Trigeminal neuralgia, Atypical odontalgia Referred pain of cardiac origin Subacute thyroiditis, Odontalgia
Cervical	Tension headache Arthritis, Eagle syndrome Carotidynia
Throat	Glossopharyngeal neuralgia Eagle syndrome Subacute thyroiditis Carotidynia

Source: B Antonia Baleivnas, Michael A Siegel and Edward G Grace: A clinical approach to the diagnosis of facial pain dental clinics of North America. 36 (4) Oct 1992. pp.993.

Orofacial neurogenic pain

Neuralgias

A neuralgia by definition is a paroxysmal, intermittent intense pain usually confined to specific nerve branches.

- Trigeminal neuralgia
- Glossopharyngeal neuralgia
- Superior laryngeal neuralgia
- Occipital neuralgia

Trigeminal neuralgia

Trigeminal neuralgia is also known as *Tic douloureux*. This is a very painful condition that involves one side of the face along the distribution of one or more divisions of the trigeminal nerve. Its etiology is not known.

Key features of the trigeminal neuralgia are as follows:

- Average age of onset is approximately 50 years
- Preponderance for females
- Pain is brief shock like and lancinating
- Shows slight higher predilection for the right side
- Pain usually is precipitated by non painful stimuli such as shaving, washing, talking or brushing the teeth
- Duration of each painful attack is of few seconds
- Frequent attacks give rise to burning sensation along the same distribution
- 2nd and 3rd division of trigeminal nerve are most commonly involved
- The pain does not cross to the opposite side of the face
- Rarely the condition may affect the face bilaterally

- Neurologic examination is essentially normal
- Local anaesthetic injection in the trigger zone temporarily eliminates pain.

Trigeminal neuralgia must be differentiated from other causes of orofacial pain; namely:

- Dental pain
- Sinus disorders
- Head and neck neoplasms causing pain
- Post-herpetic neuralgia
- Cluster headaches
- Migraine
- Atypical facial pain.

It must be remembered that central lesions such as multiple sclerosis can produce trigeminal neuralgia. Compression of trigeminal root or ganglion by tumor, aneurysms or vascular malformations can also produce symptomatic trigeminal neuralgia. When in doubt, an imaging study of the head (CT or MRI) should be done in order to distinguish symptomatic trigeminal neuralgia from the idiopathic type.

Treatment of trigeminal neuralgia

There are two treatment modalities available: medical and surgical.

Medical treatment

This involves the use of carbamazepine (Tegretol), 100 mg or 200 mg twice daily. The usual maintenance dose is 600 to 1200 mg per day in divided doses until symptoms subside or complications of medication occur.

The common side effects are drowsiness, dizziness, unsteadiness, nausea and anorexia. Rarely

complication such as aplastic anemia may ensue. Periodic complete blood cell count and liver and kidney functions tests are necessary in these patients. Complete blood cell count every two weeks during the first 2 months and quarterly thereafter is recommended. If there is a good response to medication, the dose of the medication can be tapered slowly over 4 to six weeks after the patient has been symptom free for a few months.

The other useful drug is Baclofen (Lioresal) 5 to 10 mg three times daily until patient is symptom free. The usual maintenance dose is 60 mg/day. Often side effects such as drowsiness and dizziness occur. This drug should not be withdrawn suddenly as hallucinations and seizures can occur. Drug should be tapered and stopped gradually after the patient is symptom free for about 2 months.

Other drugs in use are:

- Phenytoin (Dilantin) 300-400 mg/day
- Divalproex sodium, clonazepam and pimozide (Orap). These are less commonly used drugs.

Surgical management

It has been reported that about 30 percent of patients with trigeminal neuralgia fail medical treatment. Variety of surgical techniques have been advocated for these patients. These include:

- Alcohol block
- Alcohol gangliolysis
- Radiofrequency thermoneurolysis
- Neurectomy
- Glycerol gangliolysis

- Microvascular decompression
- Trigeminal tractotomy.

Success of surgical treatment is variable.

Glossopharyngeal neuralgia

Glossopharyngeal neuralgia affects the region supplied by the distribution of the glossopharyngeal nerve and occasionally the area supplied by the auricular nerve and pharyngeal branches of the vagus nerve. Because of the involvement of the vagus nerve the condition is also known as vagoglossopharyngeal neuralgia.

Glossopharyngeal neuralgia is less common as compared to trigeminal neuralgia.

The characteristic of pain is as follows:

- Severe, stabbing or burning pain
- Located in ear, base of the tongue, tonsillar fossa or beneath the angle of the jaw
- Pain is usually unilateral and rarely bilateral.
- Pain lasts for about 2 seconds to 2 minutes
- Pain may be provoked by swallowing, chewing, talking or yawning
- Neurologic examination does not reveal any abnormality.

The cause of glossopharyngeal neuralgia is unknown. In those with symptomatic glossopharyngeal neuralgia tumor or infection in the region may be the cause. MRI therefore needs to be carried out in order to exclude these possibilities.

Treatment of glossopharyngeal neuralgia involves Tegretol and other medications as recommended for trigeminal neuralgia. There are also certain selected surgical approaches available to treat the condition.

Superior laryngeal neuralgia

Superior laryngeal neuralgia is a rare condition.

Key features include:

- Pain is felt in the throat, submandibular region or under the ear
- Swallowing, singing loudly or head turning may trigger the pain
- Treatment includes Tegretol and neurectomy.

Occipital neuralgia

- Cause of this condition is not known. Often trauma to the nerve has been blamed
- In occipital neuralgia the pain is of jabbing nature and is felt along the distribution of the greater or lesser occipital nerves
- The affected nerve is often tender on palpation.

Treatment includes injection of local anaesthetics and corticosteroids.

Neuralgia caused by neuromas

Rarely neuromas cause pain of neuralgic nature due to pressure exerted by them on the nerves.

Post-herpetic neuralgia

Post-herpetic neuralgia is a complication of Herpes Zoster (Shingles) involving the face.

Key features include:

- Intense pain along the affected nerve
- Presence of vesicles along the affected nerve
- High doses of Acyclovir often useful in reducing the symptoms.

Post-traumatic/operative neuralgias

These conditions are characterized by neuralgic type of pain and occur as a result of trauma caused either by episodes of violent nature or operative procedures.

Central causes of orofacial pain*Anesthesia dolorosa*

This is a painful area of anesthesia or dysesthesia along the trigeminal nerve distribution which arises after damage to the nerve or ganglion. Neurosurgery in the area also may cause this condition. The condition is characterized by pain and decreased sensibility to pain and temperature on one or more divisions of trigeminal nerve.

Treatment of anesthesia dolorosa involves the use of tricyclic antidepressants.

Thalamic pain

Thalamic pain is characterized by facial pain, dysesthesia and impaired sensation to pinprick and temperature. The condition is not due to a lesion of trigeminal nerve, instead, it is due to a lesion of the thalamus. This condition is rare.

Unclassifiable orofacial pain

- Atypical Tooth Ache (Atypical odontalgia, Phantom tooth pain, Dental migraine)
- In this category patient presents himself/herself with all the typical features of an acute toothache.
- Etiology of this condition is not known
- Pain in a tooth or tooth site is severe, throbbing, continuous and is felt even on the other side by crossing the midline

- Molars are commonly involved
- Majority of these patients suffer from depression
- Chewing often aggravates pain
- Condition is common among females
- Antidepressant drugs (e.g. amitriptyline) are known to have beneficial effects in relieving pain
- Intraoral radiographs are of no direct help
- Clinically there is no observable pathology
- Diagnostic local anesthetic blocks usually are of no help
- Many a times dentists extract tooth or perform endodontic procedures unnecessarily.

It is extremely important to differentiate atypical odontalgia from toothache of pulpal origin. The following features are common to atypical odontalgia (and not to pulpitis).

- Constant pain in the tooth with no obvious source of pathology
- Heat, cold, sweets, etc. do not provoke or alter the degree of pain
- Toothache does not change its character even after months
- Repeated dental therapies have no effect
- Response to local anesthetic injection is variable and uncertain
- Tricyclic antidepressants have beneficial effect.

Orofacial pain of vascular origin

This group of pain consists of headaches such as migraine, cluster headaches and giant cell arteritis.

Migraine headaches

Migraine headaches are common. These are usually due to abnormal cerebral vasodilation.

Key features include the following:

Migraines are:

- Periodic
- Unilateral
- Associated with irritability, photophobia, nausea and vomiting
- Common in females
- Usually occur in 2nd and 3rd decades of life
- Common in family members of migraine sufferer
- Preceded by an aura usually visual (blind spot or zig-zag lines or flashing light)
- Pulsating unilateral severe headaches.

Treatment consists of:

- Use of ergot alkaloids
- Antidepressants
- Beta adrenergic blockers
- Slow calcium channel blockers.

Cluster headaches

This condition is characterized by bouts of pain in spaced clusters of paroxysms.

They are:

- Common in males
- Common in 3rd to 5th decade
- Common in smokers
- Not preceded by any aura
- Of burning quality and very severe
- Of short duration lasting for few minutes to 30 minutes
- Painful headaches felt behind the orbit unilaterally
- Associated with lacrimation.

Therapy of cluster headaches include:

- Avoidance of vasoactive foods such as chocolates, cheese, etc.
- Use of Ergot alkaloids
- Use of Corticosteroids.

Giant cell arteritis

Giant cell arteritis, also known as temporal or cranial arteritis is characterized by vasculitis of the branches of external carotid artery. In this disease the endothelium of the cranial arteries is inflamed with the appearance of giant cells when examined histologically.

Key features include:

- Is not a common disorder
- When present, usually affects older individuals
- Is very painful and of throbbing nature
- Pain is felt behind the orbital region
- Pressure on the area or lying down worsens pain
- Skin over the area is tender and erythematous
- Low grade fever may be present
- Loss of vision may be a complication caused by involvement of ophthalmic vessels.

Treatment involves urgent administration of corticosteroids.

Headaches in hypertension

Usually headaches experienced in uncontrolled hypertension are confined to the occipital region. Visual disturbances may also accompany such episodes.

Muscular pains

Pain of muscular origin is generally continuous, deep dull and often described as pressure or tightness. This is the most common cause of pain in the head and neck region. Muscular pains can be categorized as:

- Splinting pain
- Myospasm pain
- Myositis pain
- Myofascial pain and dysfunction
 - Tension type headache (TTH)
 - Coexisting migraine and TTH

Splinting pain

This is protective in function. It involves muscle tightening and occurs when part of the body is injured and requires rest. In orofacial region splinting of the masticatory muscles may be a response to increased tension, stress, clenching, bruxism, recent dental operations, occlusal interferences, local anesthesia, TMJ pain and so on.

Symptoms of splinting pain are:

- Local muscle tightness
- Pain on contraction of the involved muscle
- Feeling of muscle weakness
- Restricted muscle movement
- Tense muscle on palpation.

Management include:

- Removal of the cause
- Moist heat application
- Rest to the area
- Short course of muscle relaxants.

Myospasm pain

This is characterized by involuntary spasm of muscles due to overstretching of muscles that are previously weakened through protective “splinting”. For example, after prolonged opening of the mouth for dental procedures myospasm pain can occur. Often medications such as tranquilizer may also cause myospasm pain.

Symptoms myospasm pain includes:

- Shortened painful muscle with gross limitations of movement
- Dull pain with occasional sharp lancinating pains in the ear or temporal region
- Restricted jaw opening.

Management includes:

- Moist heat application
- Gradual stretching of muscle.

Myositis pain

This results due to inflammation of the muscle. Most commonly involved muscle in the head and neck region is masseter and medial pterygoid muscle.

Causes of myositis pain includes:

- Dental sepsis, injury, surgery or needle abscess.

Symptoms includes:

- Painful jaw opening
- Swelling of the region
- Fever and malaise if myositis is due to infection.

Management

- Moist heat
- Nonsteroidal anti-inflammatory drugs
- Antibiotics if infection is the cause.

Contraindications include: exercises and injections during the acute phase.

Myofascial pain

Myofascial pain is a regional referred pain syndrome associated with focally tender “trigger points” in a muscle. This can happen anywhere in the body. In the head and neck region myofascial pain is the most prevalent cause of painful symptoms in temporomandibular disorders.

Symptoms includes:

- Pain located about the normal muscle
- Pain may simulate headache, toothache or sinus pain
- Pain is deep, dull and aching
- Pain increases under stress and cold weather.

Management includes:

- Identification of the cause or predisposing factors and their elimination
- Patient education regarding good posture, stress agent man, etc.
- Home care with stretching
- Trigger point injections with local anesthetic (0.5% lidocaine)
- Needling with no injecting substance.

Tension type headache (TTH)

The pain of tension type headache is bilateral dull and aching and sometimes band like.

- Chronic TTH increases with age and is more common in females (M:F 2:5)
- These patients may report an increased tenderness of pericranial muscles and its attaching fascia even when the headache event is not present

- Patient may have problem with drug abuse
- Temporomandibular joint disorder may aggravate TTH
- Intraoral factors such as malocclusion may not contribute significantly although stabilizing intra oral appliances have been reported to be useful in treating TTH.

Articular pains

Articular pains in the head and neck region are those periarticular aching pains involving intrinsic lesions of the temporomandibular joint (TMJ) or cervical spine.

Three categories of temporomandibular articular pains exist:

- Internal derangement
- Inflammatory disorders of the joint
- Chronic hypomobilities
- Growth disorders.

It is sometimes possible that multiple factors may affect one joint.

Internal derangement

Internal derangements are disorders intrinsic to the temporomandibular joint. These include:

- Disc-condyl incoordination
- Anterior displacement of the intra-articular disc with reduction
- Anterior displacement of the disc without reduction (locking)
- Degenerative joint disease.

The first three disorders are due to abnormal functioning of the articular disc.

Symptoms of internal derangements are:

- Typically non painful
- Clicking, popping or locking of the joint upon opening or closing
- Difficulty in opening the jaw in the morning is often reported
- Sometimes patients need to manipulate their jaw with a resultant loud crack.

Etiology of internal derangement includes:

- External trauma
- Habitual para function such as bruxism
- Occlusal disharmony.

Degenerative joint disease is the non-inflammatory phase of arthritis.

Examination will reveal:

- Click or pop on palpation with mouth opening and closing
- Often joint locking on one side restricts the movement—as a result deviation of the jaw towards the affected side on closing occurs
- Tomograms show minimal posterior displacement of the condyle in the glenoid fossa
- Crepitus in case of degenerative joint disorders with limitation of movement of the condyle
- *Radiographic examination of the degenerative disease may have any of the following:*
 - Decreased joint space
 - Subchondral sclerosis
 - Cystic formation
 - Surface erosions
 - Osteophytes
 - Marginal lipping
 - Flat articular eminence.

Treatment includes:

- Reassurance
- Physical therapy
- Occlusal rehabilitation
- Orthoscopy/surgery in more severe cases.

Inflammatory disorders of the joint

These are painful disorders which include:

- Capsulitis
- Sinusitis
- Retrodiscitis
- Acute arthritis.

Symptoms include:

- Continuous pain over the joint
- Increase in pain by function
- Presence of swelling over the joint
- Restricted mouth opening
- History of trauma, infection, polyarthralgia, etc.
- Pain tends to wax and wane with the course of inflammation.

Signs include:

- Palpation over the joint elicit tenderness
- Tenderness on palpation from inside the external auditory meatus (retrodiscitis)
- Effusion in the joint may be present
- Biting on a tongue depressor reduces the pain by preventing full closure (retrodiscitis)
- Gross joint deformities on radiographic evaluation (inflammatory arthritis).

Therapy

Any of the following may be necessary.

- Reducing the conservative factors

- Construction of stabilizing splints
- Analgesics and anti inflammatory drugs
- Physical therapy
- Periodic intraarticular steroid injections
- Surgical intervention.

Chronic hypomobilities

Hypomobility of the mandible may be caused by masticatory muscle contracture, capsular fibrosis or ankylosis of the joint trauma, surgery and inflammation.

Growth disorders

These include developmental abnormalities, traumatic joint changes and neoplasms; radiographic evidence in these cases is quite suggestive.

Burning mouth syndrome

Burning mouth syndrome (BMS) is a disorder of obscure etiology. Patients usually complain of:

- Intraoral burning (tip and side of the tongue commonly involved)
- Dry mouth
- Thirst
- Taste and sleep disturbances
- Headache
- Increase in pain as day progresses
- Common in post menopausal women.

Examination reveals negative intraoral findings.

Pain of salivary gland origin

- Pain for salivary glands is localized

- It may increase by increased saliva production before meals. An affected gland is swollen and tender on palpation
- There may be trismus, fever and malaise (parotitis)
- In children mumps is the most common cause of salivary gland pain
- Sialolithiasis or mucous plugs, which often block the salivary gland duct opening, can cause pain in adults.

Ear pain

Middle ear diseases often cause pain in the ear and headaches.

- Middle ear infections (otitis media) and their complications such as mastoiditis are painful conditions
- Pain of acute otitis media is deep in the ear and irradiates to the temple region
- Pain is stabbing pounding and worse at night
- Diagnosis is often easy, patient should be referred to an ENT specialist for treatment.

Pain of maxillary sinus origin

With acute or chronic sinusitis, a feeling of constant dull, aching pressure or discomfort can often be felt in the premolars or molars of the affected area.

- These teeth may be tender on percussion
- Pain and tenderness over the affected sinus and increased pain when patient bends forward is typical of pain of sinus origin
- Patient may also report pain below the eyes

- Patient usually gives a history of upper respiratory infection
- Radiographs (water's view) often show air-fluid levels or thickened mucosa of the maxillary sinus
- Maxillary sinus infection is usually treated with a ten day course of amoxicillin and a 2 or 3 day use of a topical decongestant
- Referral to an ENT specialist is necessary if the treatment is non responsive to the antibiotic
- Ethmoid frontal and sphenoid sinus inflammations are also frequently the cause of pain in the head and face.

Pain of cardiac origin

It is not uncommon to see a patient in the dental clinic with tooth or jaw pain of cardiac origin. Occasionally dental pain may be the only symptom in these patients.

- Patients usually however complain of discomfort or pain in the substernal region left arm or in the neck
- It is important to rule out dental origin of pain in these patients. A local anesthetic block relieves pain of dental origin. If it fails the pain is of non-odontogenic source and cardiogenic source may be one of them
- Angina pectoris or myocardial infarctions are two common conditions, which cause tooth/jaw pain of cardiac origin.

Symptoms of cardiogenic pain includes:

- cyclic pain in the left side teeth/left maxilla or mandible

- toothache/jaw pain is increased by physical exertion
- toothache/jaw pain maybe associated with chest, arm or neck pain
- toothache is decreased with nitroglycerine tablets
- local manipulation of the tooth fails to aggravate pain
- history of previous cardiac problems (not necessarily always).

If cardiogenic pain is suspected immediate referral of the patient to a hospital based cardiologist or to emergency room of the hospital is mandatory.

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CHAPTER

6

**Differential
Diagnosis of
Swellings of
the Neck**

JA Giglio, DM Laskin

Learning objectives

After studying this chapter the student should be able to:

- Understand the necessity of establishing a differential diagnosis of neck masses
- Describe various classifications used to differentiate neck masses
- Perform a clinical evaluation of neck masses
- Establish a differential diagnosis of neck masses based on specific regions in the neck.

Introduction

A thorough physical examination of the head and neck should be included as part of the evaluation of every dental patient. Because the pathologic lesions that can arise in the neck are varied and diverse, the diagnostic thought process should be orderly and inclusive so that a reasonable differential diagnosis can be established and prompt, effective treatment rendered.

Clinical relevance

Neck masses are a common finding. They most often represent a self-limited inflammation, a benign neoplasm, or a congenital anomaly; however, these lesions can also be malignant.

Therefore, the evaluation process must be orderly and inclusive so that a proper diagnosis can be made and appropriate treatment quickly rendered.

Anatomically, the neck comprises only a small percentage of the total body area, but it contains many vital anatomic structures and diverse tissue types. Consequently, the pathologic lesions that can arise in the region are equally as varied and diverse.

Classification

Neck lesions have been classified in a number of ways. Common methods are according to:

- *Anatomic location*, i.e. median or paramedian swellings, or *tissue of origin*.
- However, lesions have also been differentiated as *primary or secondary*, or divided into *midline, lateral, and low-lying lesions*.
- Other classifications have used the *organ systems approach*, i.e. vascular, nervous, etc. or the *etiologic origin*, separating them into either inflammatory, neoplastic, or congenital categories.
- The *patient's age* has also been used as a means of grouping lesions.
- Some classifications have even included palpable *normal anatomic masses* such as the transverse process of C2, the styloid process, the mastoid tip, the greater cornu of the hyoid bone, and the thyroid cartilage.

All classifications, however varied, are useful in that they help the clinician to order the diagnostic thought process and form an initial impression of the clinical findings.

Clinical evaluation

Clinical evaluation must always begin with a *thorough history, physical examination, and close attention to symptoms*.

- Most diagnostic errors occur because of an inadequate history and clinical examination, with an over-reliance on sophisticated imaging techniques
- Patients at risk for serious clinical problems can

often be identified through findings in the history, such as progressive enlargement of the mass or prolonged hoarseness

- A careful history will also begin to narrow the range of possible lesions and guide deductive reasoning. For example, age can be a factor. In adults over 40 years of age, metastatic neoplasms are strong possibilities and should seriously be considered. For example, a non-thyroid neck mass in an older patient has an 80 percent probability of being neoplastic, with 80 percent of them being malignant
- In young adults and teenagers, inflammatory lesions, especially from a third molar pericoronitis, are common
- Infectious mononucleosis and thyroid malignancies should also be suspected in the young age group.
- Developmental lesions, as well as the more common lymph node response to infection and inflammation should be considered in children.
- The patient's report of the duration that a lesion has been present is also helpful information.

A rule of thumb states that a mass present for *seven days is inflammatory, seven months is neoplastic, and seven years is congenital*.

Because certain lesions are found in discrete anatomic locations, a knowledge of those associated with specific regions in the neck is useful in the differential diagnosis. The following discussion uses this approach.

Differential diagnosis: midline lesions

- Midline swellings of the neck (Table 6.1) are characteristically caused by such conditions as

Table 6.1: Midline swellings

- Thyroglossal duct cyst
- Epidermoid cyst
- Dermoid cyst
- Submental lymphadenitis
- Submental abscess
- Thyroid gland tumors
- Ectopic thyroid

the congenital thyroglossal duct cyst (elevates with tongue protrusion) (Fig. 6.1), the developmental epidermoid or dermoid cyst (does not elevate with tongue protrusion) (Fig. 6.2), and the neoplastic growths from the thyroid and its isthmus.

- The presence of ectopic thyroid tissue is rare, but merits consideration. This midline mass can appear at the base of the tongue in the region of the foramen caecum (lingual thyroid), more deeply situated infralingually, or high in the midline of the neck. Symptoms, when present,



Fig. 6.1: Thyroglossal duct cyst presenting as a midline swelling (Arrow)



Fig. 6.2: Epidermoid/dermoid cyst in the midline of the neck

include dysphagia, difficulty with speech, and a feeling of fullness in the throat. Before a suspected lesion is removed, care must be taken to establish that the patient has a normal functioning thyroid gland. Removal of ectopic thyroid tissue without such confirmation can result in the production of a hypothyroid or myxedematous state because it may represent the patient's only thyroid tissue.

- The submental lymph nodes drain the lip, anterior tongue, and the region of the mandibular incisors and anterior gingiva. Enlargement of these nodes (submental lymphadenitis) reflects possible pathology in these areas. An infection from a mandibular incisor can cause an abscess in the submental space (Fig. 6.3). An abscess occupying this space presents as an anterior swelling, which may be accompanied by dysphagia and dyspnea.
- Swelling of the submental space along with bilateral swelling of the submandibular and sublingual spaces is termed Ludwig's Angina, and



Fig. 6.3: Abscess in the submental space

can be life threatening unless treated aggressively.

Differential diagnosis: lateral cervical swellings

The lateral cervical swellings can be either discrete or multiple, and located high or low in the neck. Because of their variability they tend to present difficult diagnostic problems.

Discrete lateral swellings

- *High level swellings:* These lesions (Table 6.2) are very often related to the major salivary glands. The ranula is a form of mucous retention phenomenon that develops in association with either the sublingual or submandibular gland
- On occasion, the ranula herniates through the myelohyoid muscle and is subsequently referred to as a “plunging ranula” (Fig. 6.4). This lesion is palpable high in the neck
- The pleomorphic adenoma is the most common salivary gland tumor and is typically associated with the parotid gland; however, it can involve

Table 6.2: High level swellings in the neck

<i>Lesion</i>	<i>Type</i>
• Ectopic thymus	• Developmental
• Branchial cleft cyst	• Developmental
• Salivary gland tumor	• Neoplastic
• Carotid body tumor	• Neoplastic
• Cystic hygroma	• Developmental
• Neurofibroma	• Neoplastic
• Fibroma	• Neoplastic
• Hemangioma	• Neoplastic/hamartomatous
• Plunging ranula	• Developmental
• STOI	• Neoplastic
• Enlarged nodes	• Neoplastic/inflammatory
• Sialadenitis	• Inflammatory



Fig. 6.4: Plunging ranula

the submandibular gland as well. When arising in the tail of the parotid gland, the tumor will be palpable as a firm, discrete, non-fixed mass in the region of the mandibular angle

- Pleomorphic adenomas of the submandibular

gland are located in the submandibular triangle. These masses are usually freely mobile, discrete, and not adherent to the overlying skin. Malignant tumors of the submandibular gland should be suspected if the patient is an older adult, the swelling is firm and fixed, it continues to enlarge, and it is painful

- Sialoadenitis should be considered if the swelling is acute and painful (Fig. 6.5)
- Bidigital palpation will often help to define the presence of calculi within the gland or duct system. Radiographs (Fig. 6.6) and sialography will confirm the diagnosis
- It should be remembered, however, that the forming calculus may not have calcified and, consequently, may not be evident on the radio-



Fig. 6.5: Sialoadenitis of the submandibular gland

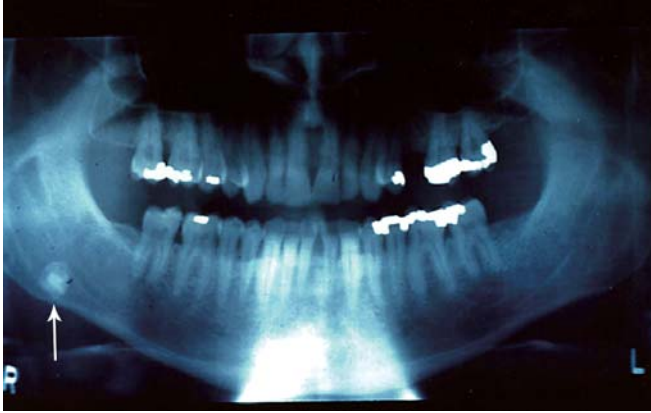


Fig. 6.6: Radiograph showing salivary stone (Arrow) of the patient shown in Fig. 6.5

graph. However, contrast medium used for the sialogram will usually not flow past such an obstruction and thus indicates its presence

- Other discrete high level lateral cervical swellings include the carotid body tumor, the branchial cleft cyst, and the cystic hygroma. The branchial cleft cyst (Fig. 6.7) is developmental in origin, grows slowly, and is generally found in adolescents and young adults. The freely mobile, painless lesion is usually located anterior to the sternocleidomastoid muscle in the upper third of the neck. Aspiration will reveal cholesterol crystals, which are diagnostic for the lesion
- Carotid body tumors are small, discrete masses located beneath the angle of the mandible at the bifurcation of the common carotid artery. They are usually asymptomatic and can be confused



Fig. 6.7: Branchial cleft cyst anterior to sternocleidomastoid muscle

with the branchial cleft cyst. However, carotid body tumors are located deeper, usually are firm, and cannot be displaced vertically as readily as the branchial cleft cyst

- The cystic hygroma (lymphangioma) is usually seen in infants and children. It is palpable as a soft mass posterior to the sternocleidomastoid muscle. The lesion is initially asymptomatic, but can enlarge to produce symptoms of pressure in the region of the trachea. It is frequently associated with karyotypic abnormalities and various malformation syndromes
- Neurofibroma, lipoma, fibroma, and hemangioma should also be included in the differential diagnosis of discrete lateral cervical swellings
- The sternocleidomastoid tumor of infancy (STOI) is relatively uncommon, presenting as a firm, well circumscribed, mass within the sternocleido-

mastoid muscle in infants 1-8 weeks of age, which may be associated with torticollis. The STOI must be considered in any infant with a lateral neck mass. It usually is a unilateral condition but, bilateral involvement does occur

- Ectopic thymus tissue is a rare lesion that develops along a line from the gonial angle of the mandible to the manubrium of the sternum. The lesion can occur in infancy, but the majority occurs between childhood and early adulthood, corresponding to the normal growth of the thymus gland. The thymus gland plays an established role in the development of immunologic competence. Therefore, when the lesion occurs in the neonate or very young patient, it is important to determine preoperatively whether the mass represents all or only part of the infant's thymus tissue.

If there is no mediastinal thymus present, only a subtotal removal of the mass should be attempted.

Low level swellings

The intrathoracic goiter and esophageal fibroma occur as low level, lateral swellings in the neck (Table 6.3). The goiter can be palpated in the root of the neck above the clavicle. It causes deviation of the trachea and ascends and descends with swallowing.

- The esophageal fibroma is a rare tumor. It too ascends and descends with swallowing and cannot be differentiated clinically from a goiter
- Firm, palpable lymph nodes in the supraclavicular region may represent metastatic carcinoma from the gastrointestinal tract. In addition, a

Table 6.3: Low level lateral cervical swellings

- | | |
|------------------------|----------------------|
| • Intrathoracic goiter | • Esophageal fibroma |
| • Metastatic carcinoma | • Sarcoidosis |

supraclavicular mass requires evaluation for generalized adenopathy and possible infra-clavicular neoplasms (e.g. lung, breast, ovary, testes, and colon), because masses in the lower neck commonly arise from pathology below the clavicle

- Sarcoidosis is a disease of unknown etiology characterized by epithelioid-cell follicular formations within the organs involved. The lungs and lymph nodes are frequent sites of involvement. Lymphadenopathy is most marked in the peribronchial region. Consequently, enlarged lymph nodes may be palpated low in the neck above the lung apices.

Multiple lateral cervical swellings

Multiple lateral neck swellings usually involve the lymphatic system.

- Enlarged, tender cervical lymph nodes are frequently seen in young children. Odontogenic infection and tonsillitis are common causes
- Frequently, following acute infection, the involved nodes will remain enlarged for extended periods. For example, cervical nodes enlarged due to infection with the gram-negative bacteria causing Cat Scratch Disease will remain enlarged from two to four months following remission of the disease. These nodes are freely moveable, occasionally

tender, but not unduly firm

- Pulmonary tuberculosis may become disseminated by either lymphatic or vascular spread. Localized tuberculous infection of the cervical lymph nodes is termed a *scrofula*. The infected nodes become swollen and palpable, and are typically tender or painful. They may abscess or remain as a granulomatous lesion
- Fixed, firm, enlarged, and painless nodes in the neck may be indicative of lymphatic spread from a primary carcinoma in the oral cavity, oro/nasal pharynx, maxillary sinus, nasal cavity, or facial region. A neck swelling may be the first clinical manifestation of such cancers. These masses are typically located behind and below the mandibular gonial angle. A thorough oral, nasal, and pharyngeal examination should be performed to rule out a primary lesion
- Enlarged, matted cervical nodes can also be indicative of a primary disease such as lymphoma, leukemia, Hodgkin's disease, or tuberculosis.

Definitive diagnosis

- Neck swellings should always be viewed with a high index of suspicion. Because of the possibility of primary tumors arising in the neck, and the frequent occurrence of metastatic lymph nodes, many clinicians consider all neck lesions as malignant until proven otherwise. This approach, while sound, should be used with tact and reassurance to the patient so as not to cause undue alarm
- Careful evaluation of data assembled from the

history, physical examination, plain radiographs, and computerized tomography (CT scan) and magnetic resonance imaging (MRI) will help establish a preliminary clinical diagnosis

- Fine needle aspiration and/or surgical biopsy will establish the definitive diagnosis and dictate the final course of treatment.

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CHAPTER

7

**Differential
Diagnosis of
Swellings of
the Face**

SR Prabhu, H Al Bayaty

Learning objectives

After studying this chapter the student should be able to:

- Know the various causes of facial swellings
- Know the steps taken for diagnosis of facial swellings
- Understand the clinical features of various swellings due to infections
- Understand the general characteristics of facial swellings due to cysts and tumors of the jawbones
- Know the key features of facial swellings due to parotid gland pathology
- Know some aspects of non-dental conditions, which may mimic facial swellings.

Introduction

Whenever a patient reports with a facial swelling the clinician should find out the nature of the swelling and its possible cause.

In this chapter the term swelling is used to mean an abnormal single or multiple, localized or diffuse enlargement(s) of the structure(s) of the face.

Some general points to remember include:

- Most facial swellings are inflammatory in origin. Among these, swellings due to physical trauma are common
- Odontogenic infections are the other common causes of facial swellings. Important among these are dento-alveolar abscesses, osteomyelitis of the jawbones and those involving fascial spaces
- Skin and salivary gland swellings particularly those of the parotid glands are commonly caused by infections
- Non-inflammatory facial swellings include developmental odontogenic cysts and odontogenic tumours. These are relatively uncommon.

- Non-odontogenic cysts and primary bone tumors are also a major cause of facial swelling. These however are less common
- Non-neoplastic and non-inflammatory primary bone diseases causing facial swelling are occasionally encountered in dental practice. Fibrous dysplasia of the jaw bone is a good example in this category
- Occasionally the clinician may be confronted with patients showing signs of facial swelling or fullness of the face that have no identifiable local causes. In such circumstances the causes may be of systemic origin such as nephrotic syndrome, myxoedema, obesity, etc.
- It must be noted that the facial swellings can present as solitary or multiple firm nodules/growths or as fluctuating fluid/pus filled masses. In most instances these present clear-cut borders. Benign tumors, chronic abscesses and cystic swellings come under this group. Facial swelling involving larger area of the face may often lack definable borders. Acute inflammatory swellings often fall in this category.

In view of the above stated reasons it is important that the clinician should be knowledgeable of diagnosing and managing these conditions with confidence.

A list of clinical entities, which belong to different categories of facial swellings, is provided in the following paragraphs. It is beyond the scope of this chapter to discuss each of the disease entity listed. The reader is advised to refer to standard textbooks on Oral Pathology and Oral and Maxillofacial Surgery

for further information. However, where appropriate, some general points that assist the clinician to make a diagnostic approach to these lesions are briefly mentioned.

Facial swelling due to physical trauma

In this category facial swellings can occur depending on the nature, site and the degree of severity of the physical trauma. Trauma may be derived from the physical assault, road traffic accident or any other traumatic episode. If the injury is mild and superficial, the damage may be confined only to the soft tissues of the face. This can give rise to facial swelling which is manageable in the dental clinic. Major assault or vehicular accidents on the other hand may result in trauma involving jawbones and the facial soft tissues. These traumatic episodes result in gross facial swelling and some times may threaten to compromise the airway. Patients with these injuries are treated in the hospitals under the care of oral and maxillofacial surgeons.

Facial swelling due to odontogenic infections

Infections from dental sources causing facial swellings are commonly encountered in dental practice. Some of these can be potentially fatal when they spread to facial spaces and cause airway obstruction. Through the regional veins an uncontrolled infection can spread to the brain and cause serious and fatal complications.

When a patient with facial swelling due to odontogenic infection reports to the dentist, the following steps must be undertaken:

History

- Duration of the infectious process
- History relating to the events leading to the swelling
- History of trauma, treatment received and
- presence of general symptoms such as fever, headache, etc.

Clinical examination

Examination should be carried out for important clues such as:

- Is the swelling localized or generalized?
- Is the swelling unilateral or bilateral?
- Is the swelling firm or fluctuant?
- Is the swelling warmer than the unaffected side?
- Is the swelling red and inflamed compared to the unaffected side?
- Is the swelling tender?
- Is the swelling pointing towards the area offering least resistance?
- Is the swelling associated with a sinus tract and opening?
- Is the swelling causing loss of function of the area?

Investigations to be carried out include:

Hematology

- White Blood Cell Count
 - Leukocytosis is present in acute infections.
- ESR estimation
 - May show a rise in non-specific infections.

Imaging

- Usually no change on plain X-ray films during the first week

- CT scan is useful in determining the extent of space infections.

Aspiration

- In anaerobic infections the pus is dark and has malodor
- White yellow pus indicates gram positive cocci
- “Sulfur” granules indicate Actinomyces organisms.

Culture and antibiotic sensitivity testing

This is of vital importance for rapidly spreading infections and in medically compromised patients. Causative microorganisms may show resistance to certain antibiotics. This fact should be kept in mind in all major infections treated in dental practice.

Blood cultures

- These are necessary when serious infections with sepsis occur.

Therapy

- Penicillin is the drug of choice
- Clindamycin, cephalosporins, erythromycins and Doxycyclines are used for those allergic to penicillin
- Metranidazole is effective against anaerobic bacteria
- Removal of the source of infection is an important step.

Most important of the odontogenic infections causing facial swelling are:

- Pericoronitis
- Periapical abscess

- Subperiosteal abscess
- Osteomyelitis and
- Fascial 'space' infections.

Briefly some key features are discussed here. For a detailed discussion the reader is referred to standard textbooks on the subject.

Facial swellings due to pericoronitis, periapical abscess (dento-alveolar abscess) and sub periosteal abscess

Generally speaking, these infections do not cause gross facial swellings. Their primary involvement is periapical alveolar bone/periosteum with or without involvement of the surrounding soft tissues. However, if the host resistance is low and virulence of the organisms is high, there can be a wider involvement including those of the fascial spaces. Under these conditions facial swelling is noticed. Classic cardinal signs of inflammation are present in these patients (Fig. 7.1A). Chronic dento-alveolar abscess when perforates the periosteum tends to be pointing on the surface of the skin (Fig. 7.1B).

Facial swelling due to osteomyelitis

Facial swellings due to acute and chronic osteomyelitis are common. In acute osteomyelitis the following features are observed:

- Pain and swelling of the jawbones is a consistent feature of acute osteomyelitis
- Infection is almost always of odontogenic in origin
- Paresthesia of the lip is frequently reported
- No radiographic changes during the first 10 days. Moth eaten appearance is seen after the second week



Figs 7.1A and B: Facial swelling on the right maxilla in a nine year old girl due to acute dento-alveolar abscess involving the roots of the upper right first molar. Note the signs of acute inflammation such as swelling and redness **(A)**. In **(B)** Chronic dento-alveolar abscess originating from lower right first molar has perforated the periosteum. The abscess tends to point out as a localised swelling. Note the relative lack of signs of inflammation
(Courtesy: Dr H Al Bayaty)

- Response to antibiotics is generally good
- Surgical intervention becomes necessary to remove and necrotic tissues.

Chronic osteomyelitis is less serious due to its chronicity. Facial swelling is present but it is not as tense as the swelling seen in acute osteomyelitis (Figs 7.2A and B).

Facial swellings due to fascial ‘space’ infections

- Fascial space infections cause extensive facial swelling due to accumulation of inflammatory exudates and tissue edema (Fig. 7.3)
- The condition is also associated with fever and toxemia



Fig. 7.2A: Facial swelling due to chronic osteomyelitis. Note the draining sinuses on the face. The discharge reveals frank pus due to non-specific infection

Fig. 7.2B: Facial swelling due to actinomycosis. Note multiple sinuses opening on the face. The discharging actinomycosis reveals “sulphur granules” containing filamentous organisms



Fig. 7.3: Three patients showing facial swelling due to acute infection of the fascial spaces

- Space infections are almost always due to odontogenic infections
- Ludwig’s angina is the most serious of the fascial infections. It can often be fatal

- Swelling of the floor of the mouth and lower part of the face is tense, tender with characteristic board-like firmness. Overlying skin is shiny
- Opening of the mouth is restricted
- Systemic symptoms are always present and severe
- Airway obstruction is often a possibility due to oedema of glottis and position of the tongue pushing soft palate
- Immediate admission to hospital and aggressive antibiotic treatment is required. Sensitivity testing and culture studies should be undertaken as early as possible. Airway obstruction should be relieved and drainage of the swelling must be carried out immediately.

Facial swelling due to cysts and tumors of the jawbones (Figs 7.4A and B)

Cysts and tumors of the jawbones are frequently encountered in dental practice. Majority of these cause facial swellings. These can be odontogenic or non-odontogenic in origin. A detailed list is as shown under:

Cysts of the jawbones

Odontogenic cysts

A. Developmental cysts

- Dentigerous cysts
- Eruption cysts*
- Odontogenic keratocyst
- Gingival (alveolar) cyst of the newborn*
- Gingival cyst of the adult*
- Lateral periodontal cyst*
- Calcifying odontogenic cyst

* Those lesions generally do not produce facial swelling. These are listed here for the sake of completeness of the classification.



Figs 7.4A and B: Examples of facial swelling due to jaw cyst and odontogenic tumor. In **(A)** a right mandibular dentigerous cyst was detected on the X-ray and confirmed histologically. In **(B)** a left mandibular ameloblastoma was suspected on the radiographic findings which was confirmed histologically. Note the absence of inflammatory signs

B. *Inflammatory cysts*

- Radicular cyst
- Residual cyst
- Paradental cyst

Non-odontogenic cysts

- Palatal cysts of the newborn*
(Epstein's pearls, Bohn's nodules)
- Nasolabial cyst
- Globulomaxillary cyst
- Nasopalatine duct cyst
- Median palatine cyst
- Median mandibular cyst
- Aneurysmal bone cyst
- Hemorrhagic bone cyst
- Static bone cyst*

* Those lesions generally do not produce facial swelling. These are listed here for the sake of completeness of the classification.

Benign and malignant tumors of the jawbones

Odontogenic tumors of the jawbone

- Ameloblastoma
- Calcifying epithelial odontogenic tumor
- Squamous odontogenic tumor
- Clear cell odontogenic tumor
- Ameloblastic fibroma
- Ameloblastic fibro odontoma
- Odonto ameloblastoma
- Adenomatoid odontogenic tumor
- Complex odontoma
- Compound odontoma
- Odontogenic fibroma
- Myxoma
- Cementoblastoma
- Periapical cemental dysplasia
- Cementifying fibroma
- Malignant ameloblastoma
- Ameloblastic carcinoma

Non-odontogenic tumors of the jawbones

- Osteoma
- Osteoid osteoma
- Osteoblastoma
- Chondroma
- Chondromyxoid fibroma
- Ossifying fibroma
- Fibrosarcoma
- Osteosarcoma
- Chondrosarcoma
- Ewing's sarcoma
- Burkitt's lymphoma

- Neuroectodermal tumor of infancy
- Regional spread or invasion by intraoral malignancies
- Metastatic tumors from kidney, prostate, colon, thyroid, breast, lung, etc.

Key features

- Pain is usually not a prominent feature of odontogenic and non-odontogenic cysts and tumors unless these lesions are secondarily infected or if the tumor is invasive or neural in origin
- Generally, cysts and benign tumors of both odontogenic and non-odontogenic origin grow slowly. When faster growth is observed, locally aggressive or a malignant lesion is suspected.
- Majority of the cysts and tumors which have grown to a considerable size cause loss of function of the jaws
- Sensory and motor changes along the distribution of the trigeminal nerve may also be observed in these patients. This depends on the location and size of the cyst or the tumor and also depends on whether the growth is infected, aggressive or malignant
- Some large lesions of the jawbone may cause pathologic fractures and cause both loss of function and sensory or motor changes
- Maxillary swellings can exert pressure on the nasal septum and cause deviation of the nasal septum, nasal stuffiness and other nasal and ear problems.

- Oral cancer (oral squamous cell carcinoma) originating in the intraoral soft tissues can invade the bone and also extend in to the cheek causing facial swelling
- Some facial swellings due to malignancy have characteristic mal odour.

Radiographic features

Radiographic features of some of the cysts and tumours are significant.

Jaw cysts and tumors/tumor-like growths of the jaw bones do present unilocular or multilocular radiographic appearances. Those presenting as multilocular radiolucencies often present diagnostic difficulties. These include:

- Ameloblastoma
- Odontogenic keratocyst
- Central giant cell granuloma
- Ameloblastic fibroma
- Odontogenic myxoma
- Calcifying epithelial odontogenic tumor
- Central hemangioma
- Aneurysmal bone cyst
- Cherubism
- Brown tumor of hyperparathyroidism.

Radiolucencies with poorly defined borders are suggestive of:

- Malignant tumors
- Osteomyelitis
- Traumatic bone cyst and
- Hematopoietic bone marrow defects.

Facial swelling caused by bone pathology involving the jawbones

Although infrequent, when primary bone diseases/disorders involve the jawbones, they may cause swelling of the face. Usually most of these lesions are painless and slow growing. Fibrous dysplasia of the jawbone is often self-limiting and may stop growing when the patient's skeletal growth stops (Fig. 7.5).



Fig. 7.5: Maxillary swelling in this young lady is due to fibrous dysplasia. Note the absence of signs of inflammation

Fibro-osseous/giant cell lesions, which cause facial swelling, are listed below:

Fibro-osseous/giant cell lesions of the jawbones

- Fibrous dysplasia
- Periapical cemento-osseous dysplasia
- Ossifying fibroma
- Giant cell tumor

- Central giant cell granuloma
- Brown tumor of hyperparathyroidism
- Cherubism
- Histiocytosis X.

Facial swelling due to salivary gland diseases: Parotid gland diseases

Most salivary gland diseases particularly those of the parotid glands cause facial swelling. These include non-neoplastic and neoplastic diseases. Important among these include the following:

- Acute and chronic parotitis
- Sialolithiasis/Sialadenitis
- Sjögren's syndrome
- Chronic alcoholism
- Salivary gland cysts
- Obesity
- Lymphoepithelial lesion
- Malnutrition
- Benign and malignant neoplasms of the parotid gland namely:
 - Pleomorphic adenoma
 - Warthin's tumor
 - Basal cell adenoma
 - Mucoepidermoid carcinoma
 - Acinic cell carcinoma
 - Adenoid cystic carcinoma
 - Adenocarcinoma.

Key Features

Important features of salivary gland swellings include the following:

- Salivary gland swellings associated with eating suggest an obstruction of the duct.

- Persistent salivary gland swelling may indicate neoplastic process or other causes such as Sjögren's syndrome, diabetes, alcoholism, etc
- Salivary gland swellings may be unilateral or bilateral. When unilateral, the cause may be of infectious origin, salivary stone causing ductal obstruction (sialolithiasis) or neoplasms. When swelling is bilateral, infection such as mumps or endocrine dysfunction may be the cause
- Pain is a variable feature. If related to eating, obstruction of the duct is suspected. If pain is not related to eating and is persistent, it could be due to infection or inflammation of the gland.
- Painless swellings may be due to neoplasms or diabetes and alcoholism
- Xerostomia is present in Sjögren's syndrome, which also can show parotid swellings.

Examination

Examination of salivary gland mainly includes inspection, palpation and sialography.

- A large swelling of long duration is usually due to a tumor (Fig. 7.6)
- A firm salivary gland swelling is usually due to a tumor
- Diffuse enlargement of a single gland suggests chronic inflammation
- Enlargement of multiple glands suggests Sjögren's syndrome, diabetes and alcoholism
- Neurologic findings are common in malignancies of the parotid gland. Facial, glossopharyngeal, vagus and hypoglossal nerves may be affected.
- Turbid saliva suggests infection.



Fig. 7.6: Examples of tumors of the parotid salivary gland. These may be benign or malignant

Radiography

- Plain views, panoramic views lateral oblique views and anteroposterior views are useful for diagnosis of salivary gland disorders.

Sialography

This is a useful test for a range of salivary gland disorders. Condition in which sialography is useful includes:

- Obstruction to the duct
- Tumors of salivary glands
- Secondary and recurrent gland infection
- Sjögren's syndrome.

Computed tomography

For non-inflammatory enlargement of salivary gland CT scan is very useful.

Other useful tests include:

- Magnetic resonance imaging (MRI)
- Radionuclide scan
- Ultrasound
- Biopsy
- Sialochemistry.

Miscellaneous causes of facial swellings

“Swollen” face due to fat deposition

Subcutaneous buccal pad of fat in an obese person can give the impression of a “swollen” face. Clinician should be able to distinguish the fullness of the face from a swollen face.

Moon face in Cushing’s syndrome

- Cushing’s syndrome is characterized by hypersecretion of Adrenocorticotrophic hormone (ACTH) which can occur due to hyperplasia of the adrenal cortex. In this condition the face is full, round and puffy. The facies is called ‘Moon’s’ face.

Swollen face due to non-inflammatory edema

In conditions where there is pathological deposition of fluids resulting in edema, face shows a swollen appearance. In nephrotic syndrome for example edema is first seen around the eyes.

Puffy face of myxoedema

In patients with severe hypothyroidism or myxoedema, edema of the face is seen around the eyes. This does not pit with pressure.

Parotid gland enlargement in systemic disease

As mentioned earlier, in diabetes, liver cirrhosis and obesity chronic bilateral enlargement of parotid glands is often seen.



Fig. 7.7: An example of angioedema of the upper lip. This can result from food allergy or orofacial granulomatosis

Allergic edema of the face

Increased capillary permeability occurs in allergic conditions.

Angioedema of the lips is a good example (Fig. 7.7).

Swellings of the facial skin

These include a wide range of conditions. Some are inflammatory and others may be cystic or neoplastic. Some examples are listed here:

- Boil, carbuncle

- Sebaceous cysts
- Epidermoid cysts
- Benign and malignant tumors of tissues of the skin.

Benign and malignant tumors of the oral tissues

- Metastatic tumors
- Angioedema
- Hematoma
- Lymphoma/Hodgkin's disease involving.

Dental clinician should be able to differentiate these conditions from those arising solely from odontogenic sources and take appropriate steps to diagnose where relevant and refer the patients to specialists for treatment.

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CHAPTER

8

Differential Diagnosis of Non-Neoplastic Swellings of the Intraoral Soft Tissues

DF Wilson, SR Prabhu

This chapter has been reproduced in part from SR Prabhu et al.
Oral Disease in the Topics: Oxford University Press, 1992.

Learning objectives

After studying this chapter the students should be able to:

- Classify intraoral non-neoplastic swellings
- Know the causes of generalized gingival enlargements
- Discuss clinical features of generalized gingival enlargements
- List localized hyperplastic lesions of the oral mucosa and discuss the features, which differentiate one from the other
- Discuss diagnostic and management aspects of generalized and localized non-neoplastic swellings of the oral mucosa.

Introduction

Intraoral swellings are common. Those presenting as localized lumps or swellings in the mouth, have to be differentiated from neoplastic lumps or swellings. Some lesions present problems in terms of their management. In majority of cases etiology of non-neoplastic lumps occurring in the mouth is inflammatory in origin. Developmental abnormalities also can cause intraoral swellings in some instances. Detailed description of lesions listed in this chapter are to be found in standard textbooks of Oral Pathology and Oral and Maxillofacial Surgery.

In the following paragraphs intraoral non-neoplastic swellings are discussed under the following headings:

- Gingival enlargements
- Developmental gingival enlargements
- Drug-induced gingival enlargements
- Inflammatory gingival enlargements
- Leukemic gingival enlargement.

Denture associated with mucosal enlargements

- Denture hyperplasia
- Papillary hyperplasia of the palate.

Localised hyperplastic lesions of the oral mucosa

- Fibroepithelial polyp
- Pyogenic granuloma
- Pregnancy tumor
- Peripheral giant cell granuloma
- Calcifying fibroblastic granuloma
- Giant cell fibroma
- Lymphoid hyperplasia.

Miscellaneous lesions

- Traumatic neuroma
- Draining sinus from periapical/periodontal pathology
- Swellings due to salivary gland disease
- Swelling due to infectious and parasitic organisms.

Brief discussion on these is given in the following paragraphs.

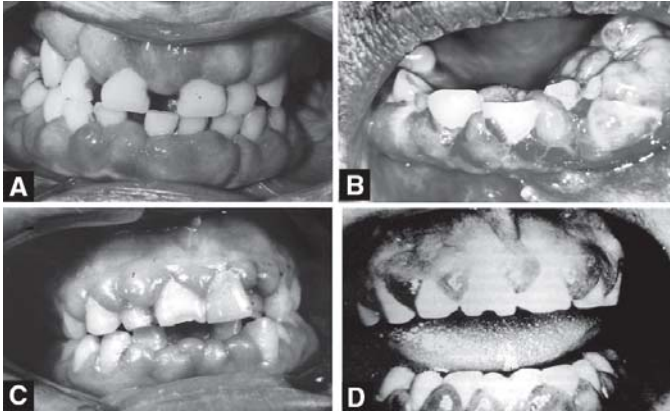
Gingival enlargements

Gingival enlargement resulting in clinically evident generalized enlargement of interdental papillae and attached gingival mucosa may be of developmental or acquired origin.

Developmental gingival enlargement

Developmental (congenital) forms of generalized gingival enlargement are sometimes referred to as 'fibromatosis gingivae' and may have a known genetic

(hereditary) basis or be of an idiopathic nature (Fig. 8.1A).



Figs 8.1A to D: **A.** Fibromatosis gingivae, **B.** Drug-induced (cyclosporine) gingival hyperplasia, **C.** Inflammatory gingival hyperplasia and **D.** Leukemic infiltration resulting in gingival enlargement

- Most cases occur solely as a gingival condition. However, fibromatosis gingivae can also be one of the manifestations of a number of rare syndromes
- Congenital/idiopathic gingival fibromatosis will generally begin to express clinical signs during childhood when teeth are erupting
- Gingival enlargement may partially or fully cover tooth crowns
- Surgical excision of excess tissue may be successful in the short-term and multiple treatments are generally required since recurrence is common.

Drug-induced gingival enlargement

- The best known example of drug-induced gingival hyperplasia is that which occurs in some patients who use the anticonvulsant drug diphenylhydantoin (Dilantin sodium) for the control of epilepsy
- Dilantin-induced hyperplasia of alveolar mucosa appears to occur most commonly in dentate patients
- The onset of Dilantin induced hyperplasia condition is generally heralded by the gradual enlargement of one or more interdental papillae. Untreated, the condition may progress until there is gross, generalized gingival enlargement. Clinically, the involved mucosa is firm and exhibits normal colouration and prominent, coarse stippling. Signs of secondary inflammation (redness, edema, hemorrhage) may be present around the teeth depending upon the patient's periodontal and oral hygiene status
- Gingival hyperplasia in patients being treated with the immunosuppressive drug *cyclosporine A* have also been reported by several clinicians (Fig. 8.1B). The clinical and histopathological features of cyclosporin A-induced gingival hyperplasia have been described as essentially similar to those of Dilantin sodium-induced hyperplasia
- Cases of drug-induced gingival hyperplasia have also been described in patients taking the drugs *nifedipine* and *diltiazem* for control of cardiovascular disorders. Clinical features of these cases are similar to other forms of drug-induced gingival hyperplasia.

Inflammatory gingival enlargement

Inflammatory gingival hyperplasia resulting in gingival enlargement usually involving the anterior segments of the jaws (Fig. 8.1C), is generally regarded as a condition caused by local 'irritation' due to the factors (microbial plaque) generally acknowledged as the cause of gingival inflammation.

- Inflammatory gingival hyperplasia may be seen more commonly in patients who are mouth-breathers
- Inflammatory enlargement of gingiva may occur in an endocrine imbalance due to puberty or pregnancy
- Nutritional imbalances may occur during pregnancy may also contribute to this condition in susceptible patients
- Vitamin C deficiency is a well-known cause of inflammatory gingival hyperplasia
- Clinically involved gingival tissues exhibit redness, oedema with loss of stippling, spontaneous bleeding, or bleeding upon probing and fibrous enlargement. The latter may involve only one or two papillae or involve more extensive areas of gingivae.

Gingival enlargement in leukemia

One of the early findings in patients with leukemia may be generalized gingival enlargement with clinical fractures identical to inflammatory gingival hyperplasia (Fig. 8.1D).

Both acute and chronic leukemia may present in this way and in either early or late stage disease. In patients who are undiagnosed leukemics features

that might alert a dentist to the possibility that an apparent inflammatory gingivitis is due to a leukemic infiltrate include:

- Rapid onset of the condition in an otherwise healthy patient with good oral hygiene
- The diffuse extent of the apparent gingivitis
- Relatively constant, spontaneous bleeding from the gingival crevices
- Pallor of the skin and mucosa
- Pallor of the conjunctiva
- Ecchymoses and petechiae of the skin/mucosa in advanced cases.

If leukemic gingival pathology is suspected in a patient then they should be sent either directly, or, through their physician for a complete blood analysis.

Denture-associated mucosal enlargements

Denture hyperplasia (epulis fissuratum, inflammatory fibrous hyperplasia)

Hyperplasia of the oral mucosa in response to irritation by ill-fitting dental prostheses occurs commonly in the adult population. The hyperplastic tissue is usually located at the periphery of the denture (Fig. 8.2), but may occur in any region where chronic physical irritation is present.

- The clinical presentation of such lesions is generally in the form of elongated folds of tissue emanating from the base of the alveolar ridge and extending into the mucobuccal or mucolabial folds into which the flange of the denture fits
- Hyperplastic tissue in these situations develops slowly, has a flabby and often fissured appearance, and is firm on palpation



Fig. 8.2: Denture hyperplasia. Folds of excess tissue (Arrow) is seen in the sulcus

- The lesion itself is usually of normal color, but in the base of the folds ulceration and inflammation may be evident.

Since the hyperplastic tissue does not provide a proper anatomical base for dentures, treatment of this condition should comprise excision of the hyperplastic tissue and fabrication of new dentures.

Papillary hyperplasia of the palate

This condition is distinctive in several ways. It occurs most commonly on the palatal mucosa and rarely in other sites such as the alveolar mucosa.

- The lesions occur most commonly on the hard palate in edentulous patients
- Papillary hyperplasia consists of numerous papillary elevations of the palate mucosa
- In most cases the lesions are red and quite

erythematous and soft. If the inflammation is less severe, the lesions are pale in color

- Microorganisms, notably *Candida albicans*, are frequently plentiful in the interpapillary spaces. However, it should be noted that elimination of the organisms using anti-fungal therapy is not always followed by regression of the lesions
- Considerable improvement in this condition can be gained by appropriate use of denture liners that need to be frequently renewed in the first few weeks of treatment. If the tissue reductions obtained in this way are inadequate, some surgical excision of the hyperplasia may be indicated before new dentures are constructed.

Localized hyperplastic lesions of the oral mucosa

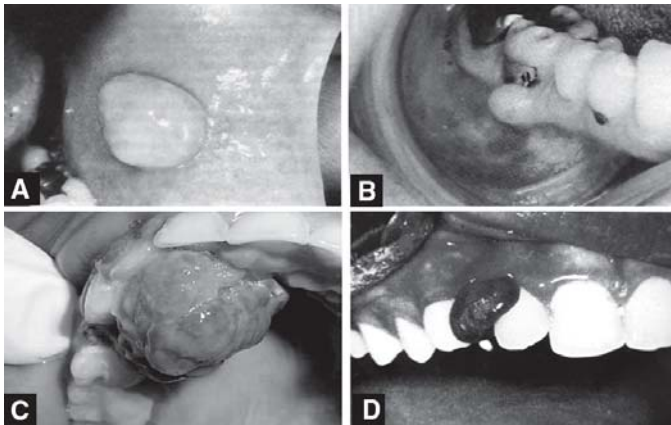
Localized hyperplastic lesions, predominantly of connective tissue origin, occur with some frequency on the oral mucosa. Majority of these lesions clinically present as localized tumor-like swellings and as such may be confused with other forms of pathology.

Fibroepithelial polyp (fibrous polyp; irritation fibroma)

Fibroepithelial polyps are the most common of the focal hyperplastic lesions involving the oral mucosa.

- They may be encountered at any site in the mouth but are most common on trauma-prone sites such as the buccal mucosa (Fig. 8.3A) inner aspect of the lip, and the tongue
- There is no specific age or sex predilection for these lesions but the majority is diagnosed in adults.

- A fibroepithelial polyp may be encountered on the gingivae in which case it usually referred to as a fibrous epulis (Fig. 8.3B)
- Clinically, fibroepithelial polyps usually present as sessile, exophytic lesions seldom more than 10 mm in diameter. Occasionally, such lesions may be pedunculated
- A flattened, pedunculated fibroepithelial polyp may sometimes be encountered on the palate mucosa underneath a full upper denture in an edentulous patient
- Fibroepithelial polyps generally exhibit a smooth surface and a color similar to normal mucosa
- Fibroepithelial polyps may exhibit a pebbled verrucous surface morphology and, if traumatized, clinical signs of inflammation



Figs 8.3A to D: **A.** Fibroepithelial polyp on the buccal mucosa, **B.** Fibrous epulis, **C.** Pregnancy tumor (Histologically diagnosable as pyogenic granuloma) and **D.** Peripheral giant cell granuloma

- The treatment of fibroepithelial polyps regardless of site is surgical excision. Recurrence is unusual except in occasional gingival lesions, which possibly arise from superficial periodontal ligament connective tissue, rather than from gingival connective tissue.

Pyogenic granuloma

- The pyogenic granuloma is a common lesion that usually occurs on the labial gingival, although it may occur anywhere in the mouth
- The lesions may be either pedunculated or sessile and the surface may be either smooth or lobulated.
- The history of the lesion is generally one of short duration and rapid growth
- The maximum size attained is about 1 cm in diameter but most lesions are smaller than this when they are first seen
- The surface of the lesion is redder than the surrounding mucosa because of its marked vascularity
- The pyogenic granuloma commonly has areas of ulceration on its surface and, for this reason, it is often quite tender on probing and tends to bleed easily
- If the lesion remains untreated, there is a gradual obliteration of the many vascular spaces, and it becomes a more fibrous lesion that clinically resembles a fibroepithelial polyp
- Pyogenic granulomas occur in patients at any age and either sexes although they tend to be more common in females and in patients during puberty/pregnancy

- The etiology of true pyogenic granuloma (as distinct from exuberant granulation tissue) is not known. For intra oral lesions it is generally accepted that local irritative factors, perhaps modified by systemic factors such as hormonal imbalances, play a role in the causation of pyogenic granuloma
- Pyogenic granulomas are essentially vascular lesions resulting from proliferation of endothelial cells. As such they are considered by some to be closely related to true hemangiomas and are occasionally referred to as 'proliferative angiomas'.

Pregnancy tumor (pregnancy epulis)

The term 'pregnancy tumor' describes the occurrence of a localized gingival proliferation in a pregnant woman (Fig. 8.3C). Most pregnancy tumors are clinically and histologically diagnosable as pyogenic granuloma.

- The lesion may develop in a small percentage of women with pregnancy gingivitis during the third month of pregnancy or sometimes later
- The lesion gradually increases in size and after childbirth may or may not regress
- It occurs as a result of minor local irritation during a period in which the tissue response is exaggerated by the endocrine adjustments, which occur during pregnancy.

Residual lesions of this nature are most effectively removed after delivery, since those removed during pregnancy have a tendency to recur.

Peripheral giant cell granuloma

(peripheral giant cell reparative granuloma, giant cell epulis)

Peripheral giant cell granulomas generally present as single, pedunculated or sessile localized swelling, reddish-blue in color, with a smooth, sometimes lobulated or ulcerated surface (Fig. 8.3D). Their occurrence is confined to the gingivae or the alveolar mucosa.

- Peripheral giant cell granulomas are seldom located posterior to the premolar teeth and vary widely in size, but are commonly between 1.0 and 1.5 cm in diameter at the time of diagnosis
- The lesions may occur in very young children and in both dentate and edentulous elderly adults, affecting females twice as frequently as males
- The nature of etiological agents responsible for the genesis of this lesion have not been established although it is generally accepted that trauma is a likely causative factor
- Treatment of a peripheral giant cell granuloma is by careful excision of the lesion. Incomplete removal generally results in recurrence
- Prior to excision it is necessary to take appropriate radiographs to rule out the possibility that the lesion is not in fact a perforating giant cell lesion of central origin and/or to determine the relationship of the lesion to adjacent teeth and bone
- In some instances where the lesion clearly involves adjacent teeth, or where recurrence has occurred due to an inability to completely excise the lesion, extraction of a tooth or teeth along with excision

of the lesion may have to be considered. Where there is evidence of underlying cortical bone involvement treatment should include appropriate surgery of involved bone.

Calcifying fibroblastic granuloma

The calcifying fibroblastic granuloma is a localized, hyperplastic lesion of the oral mucosa. Its occurrence is confined to the gingivae (i.e. the tooth-bearing areas of the jaws). It has been described under a variety of names including peripheral ossifying fibroma, peripheral cementifying fibroma, peripheral fibroma with calcification, and calcifying epulis. The calcifying fibroblastic granuloma should not be confused with the peripheral odontogenic fibroma.

- Clinically the calcifying fibroblastic granuloma presents with a history of slow or rapid growth and, at the time of diagnosis, ranges in size from a few millimeters up to about one centimeter across its base
- Small lesions may be seen to arise on the interdental papilla and/or in association with the free gingiva
- The clinical appearances of the lesion are generally unremarkable. Careful examination may reveal superficial ulceration of the lesion but without significant secondary clinical inflammatory changes
- Calcifying fibroblastic granulomas occur in females and males of any age. However, the majority of lesions are diagnosed in patients under 40 years of age.

- Treatment of calcifying fibroblastic granuloma consists of careful surgical excision. Recurrences are common.

Giant cell fibroma

The giant cell fibroma is a well-described benign, hyperplastic lesion of the oral mucosa. The lesion most commonly occurs in the gingival.

- Giant cell fibroma shows no particular sex predilection and can be diagnosed in patients of any age although most reported series indicate the lesions are more common in younger patients
- Clinically, giant cell fibroma usually presents as a small, 2-10 mm diameter, slightly warty, nodular lesion of long duration. The lesion presents normal coloration
- The treatment of choice for giant cell fibroma is surgical excision.

Lymphoid hyperplasia

Lymphoid tissues are widely distributed in the oral mucous membranes and smaller lymphoid aggregations are present in the connective tissue of the vestibular mucosa, the posterior part of the hard palate, and the lateral aspects of the tongue in the posterior region (Fig. 8.4).

- In the above named sites the lymphoid tissues may be subjected to persistent irritation and undergo hyperplastic enlargement which can present problems in diagnosis and management. Such lesions have no specific clinical features and may present simply as a focal enlargement of the affected area of the mucosa.

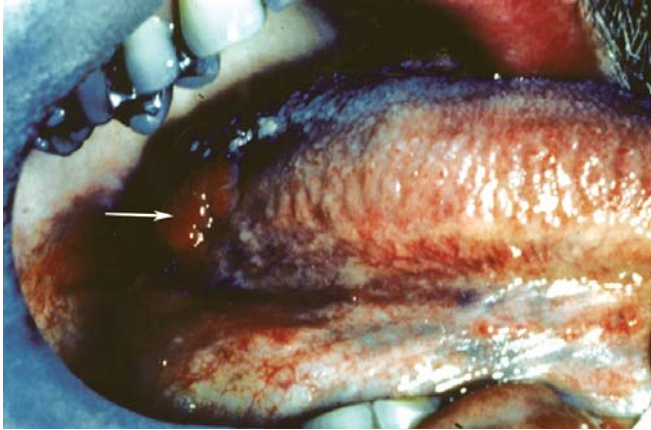


Fig. 8.4: Lymphoid hyperplasia on the postero-lateral border of the tongue (Arrow)

- The lesion described as benign lymphoid hyperplasia can present diagnostic problems to the histopathologist in terms of distinguishing this entity from malignant lymphoma. However, once a diagnosis of benign lymphoid hyperplasia is confirmed, these lesions appear to respond well to local excision or may spontaneously regress.

Miscellaneous lesions

Traumatic neuroma

Traumatic neuromas are rare hyperplastic lesions which arise following severe trauma to an area of a peripheral nerve.

- The most common oral mucosal site of occurrence is probably the mental foramen area where damage to the mental nerve may be caused during tooth extraction or other surgery

- Traumatic neuromas present as focal mucosal nodules that may be painful when pressed upon.
- Microscopically, traumatic neuromas consist of irregularly arranged and variously sized nerve bundles and fibres
- Treatment is by surgical excision.

Verruciform xanthoma

This is a rare lesion of undetermined origin. The lesion usually presents as a flat velvety, pebbly lesion of the mucosa.

The treatment of choice for verruciform xanthoma is excision.

Draining sinus from periapical and periodontal lesions

On occasion, small, focal swellings representing the mucosal extension of draining sinuses from periapical abscesses, or, deep periodontal abscesses may be seen on the gingivae. Such lesions are referred to as a parulis or “gum boil”. They are usually a few millimeters in size, inflamed and have a yellow central area that is sub-epithelial pus. Occasionally the lesions will spontaneously be exuding pus at the time of presentation. Management requires the correct identification of the tooth associated with the fistula by appropriate examination, vitality testing and radiographs.

Swellings due to salivary gland disease

Range of salivary gland lesions ranging from salivary cysts to benign and malignant neoplasms clinically present as focal, soft tissue swellings in the mouth.

Recognition of the possibility that a soft tissue swelling may be due to salivary gland disease, particularly that arising from the minor salivary glands is important. Small mucocele (mucous retention or extravasation cysts).

Ranula, swellings due to sub mandibular/sub lingual gland salivary stone, etc. are some examples of this category.

Mucocele

Retention mucocele is usually due to obstruction of minor salivary gland duct, which present clinically as gradually enlarging painless swelling commonly on the inner side of the lower lip (Fig. 8.5).

The covering mucosa is either of normal colour or may take a bluish hue. This swelling is characterized by repeated episodes of filling and rupture.

The other variant of mucocele results due to a cut in the duct system leading to accumulation of saliva in



Fig. 8.5: Mucocele of the lower lip

the interstitial tissues and this is known as *extravasation mucocele*.

Ranula refers to a swelling in the floor of the mouth. However, the majority of ranulas are retention mucoceles. Regardless of the type of the mucocele, treatment is usually by surgical removal of the swelling with the affected glands. However, recurrence is not uncommon.

Salivary stone (sialolith)

The classical feature of ductal obstruction (by means of a salivary stone) is a recurrent painful swelling in the affected gland (usually the submandibular) associated with food intake or salivary stimulation that disappears after 1-2 hours. Chronic bacterial sialadenitis is a common finding in cases of sialolithiasis.

Provoking the glandular swelling following salivary stimulation is diagnostic. Presence of a relatively large stone in the distal part of the duct or near the orifice can be palpated manually, and can be visualized radiographically (e.g. true lower occlusal radiograph in case of submandibular gland). However, a mucous plug or stenosis cannot be seen by a plain radiograph, therefore, a sialogram is indicated in such cases.

Swellings due to infective and parasitic organisms

A range of infectious organisms can produce focal soft tissue lesions in the mouth. Biopsy, culture and/or a range of other special tests are usually required to confirm a diagnosis.

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CHAPTER

9

**Differential
Diagnosis of
White Lesions
of the Oral
Mucosa**

SR Prabhu

Learning objectives

After studying this chapter the student should be able to:

- Understand the basic mechanism involved in the causation of oral white lesions
- List common white lesions that occur on the oral mucosa
- Know various investigative methods that are employed in the diagnosis of oral white lesions.

Introduction

General considerations

Some general features of oral white lesions include the following:

- White lesions of the oral mucosa are commonly encountered in dental practice
- Oral white lesions may be of a variable size ranging from a few millimeters to over several centimeters and may be single or multiple
- Some white lesions exhibit increased amount of surface keratin while others can possess necrotic epithelial surface
- Clinically, white lesions can present as patches either with smooth, rough, raised or corrugated surfaces. They may also present as pin pointed elevations or wart like growths
- A small group of white lesions are easily removable from the oral mucosa by gentle scraping (using a wet wooden spatula or sterile gauze sponge) while others are firmly adherent to the underlying structures
- Some white lesions have a genetic background with regard to their origin while most are associated with acquired causes
- A small proportion of white lesions carry increased risk of malignant transformation

- It is also possible that at the time of diagnosis some of these lesions may be frankly malignant.
- Most chronic white lesions may be asymptomatic and often detected by the dentist on routine oral examination
- Oral white lesions can be clinically seen in different shades such as paper white, ivory white, pearl white, yellowish-white, pale, blanched, cloudy or of ground glass appearance
- Their shapes and surface characteristics can vary from linear to oval, reticular, lace like, cauliflower like, cobblestone, corrugated, pinpointed, map-like, hairy and so on.
- White lesions may result in response to acute or chronic trauma of different sources including infection.

Information given in this chapter is very basic. The reader is therefore encouraged to refer to textbooks of Oral Medicine or Oral Pathology.

Clinical considerations

Clinically white lesions of the oral mucosa can be broadly categorized into two groups:

- Those which are removable easily by gentle scraping by wooden spatula and
- Those, which are not easily removable.

The former category of lesions are loosely adherent to the oral mucosa either as necrotic slough or deposits of material from external sources. Those that are non removable by gentle scraping are firmly adherent to the underlying tissues. These develop primarily because of the excessive formation of keratin.

Important steps that are to be taken in arriving at a clinical diagnosis of Oral White lesions are:

1. *A thorough history that includes:*
 - Duration of the lesion
 - Symptoms such as pain/altered taste, etc.
 - Any accompanying local or general conditions.
 - Recent changes in the lesion's size or shape.
 - Medication
 - Family History
 - Social history such as the use of tobacco, alcohol, etc.
2. *Clinical examination of the lesion:*

Color: Is it:

 - Bright white? Milky white, Snow white?, Paper white? (different shades with not much of a difference between them), cloudy?, opaque? Etc

White lesion associated with redness or pigmentations,

Shape/ Size/ Number/ Location/ Symmetry of the lesion: Is it:

 - Localized/generalized in presentation

Surface characteristics of the lesion(s): Is it:

 - Raised, flat, lace-like, patchy, papillary, cauliflower like, etc.
3. *Examination of cervical lymph nodes:*
 - Enlarged/tender/unilateral/bilateral/movable/fixed, etc.
4. *Examination of dentition:*
 - Sharp cusps, evidence of bruxism, etc.
5. *Examination of dental appliances:*
 - Sharp edges, clasps in contact with the mucosa.

- Record all information and findings on patient's record
- Draw the lesions to scale
- Obtain clinical photographs and compare the lesional characteristics at every visit
- Determine whether the lesion is removable by gently scraping with gauze sponge or wooden spatula.

Once you have carried out the above listed steps, you will proceed further to diagnose the white lesion in question.

Diagnosis of white lesions that are removable by gentle scraping

These include:

1. *Materia Alba*
 2. White furred tongue
 3. Chemical burn
 4. Thermal burn
 5. Pseudomembranous candidiasis.
1. *Materia Alba* is a white film that covers the gums. It is often seen in those who do not maintain oral hygiene. This film is generally removable on gentle scraping.
 2. *White furred tongue*: This is common in heavy smokers and in persons with systemic pathology. The tongue often becomes coated with debris and desquamating cells. Children with fevers often show furred tongue. Fur is removable by gentle scraping.
 3. *Chemical burn*: Aspirin or excessive use by the patient of clove oil placed in contact with mucosa in the buccal sulcus (in relation to a painful tooth)

can cause white slough. This can be removed by gentle scraping. History is suggestive in these patients.

Occasionally chemicals used in dental treatment procedures (e.g. Orthophosphoric acid etchant gel) can also produce chemical burns.

4. *Thermal burn*: Hot and sticky food often causes thermal burn of the mucosa. Pizza burn on the palate is a good example of thermal burn resulting in a removable white slough. History is suggestive.
5. *Pseudomembranous candidosis*: This is a condition caused by the fungus *Candida albicans*. Occasionally Erythematous form of Candidosis can also show white specks of candidal colonies on the red surface of the lesion.

Key features Pseudomembranous Candidosis include:

- Deposits of fungal colonies, epithelial cells and fibrin resulting in white lesions
- Characterized by creamy white/yellow patches/specks covering the buccal mucosa, the tongue or the palate (Fig. 9.1)
- White lesions can be removed easily by gentle scraping with a tongue spatula
- Scraped areas leave an erythematous surface with pinpoint bleeding spots
- Sore mouth, loss of taste are common complaints of these patients
- Predisposing factors for pseudomembranous candidosis include: poor denture hygiene, undiagnosed or uncontrolled diabetes, endocrine disorders, HIV/AIDS, prolonged use of antibiotics/steroids, and immunosuppressive agents



Fig. 9.1: Pseudomembranous candidosis. Note white lesions on the palatal mucosa and the tongue. These are easily scrapable

- Diagnosis is often arrived at on clinical grounds alone.
- Confirmatory tests include:
 - PAS stain; Potassium hydroxide smear and culture studies.

Diagnosis of white lesions that cannot be easily removed by gentle scraping

Lesions include:

1. Leukoedema
2. Fordyce's spots
3. Frictional keratosis
4. Smoker's keratosis
5. Chronic hyperplastic candidiasis (Candidal leukoplakia)
6. Leukoplakia
7. Oral hairy leukoplakia
8. Syphilitic leukoplakia
9. Lichen planus

10. Lupus erythematosus
11. Squamous cell carcinoma
12. White sponge nevus
13. Dyskeratosis follicularis
14. Submucous fibrosis
15. Actinic keratosis

Leukoedema (Fig. 9.2)

Leukoedema appears as a cloudy white film commonly seen bilaterally on buccal mucosa of smokers of non-caucasian descent. Whiteness disappears on digital pressure and returns after the pressure is released. This is not considered as a pathological condition.

Fordyce's spots (granules)

These are commonly seen on the posterior part of the buccal mucosa and are often bilateral. Vermilion borders of the lips are also often involved. These

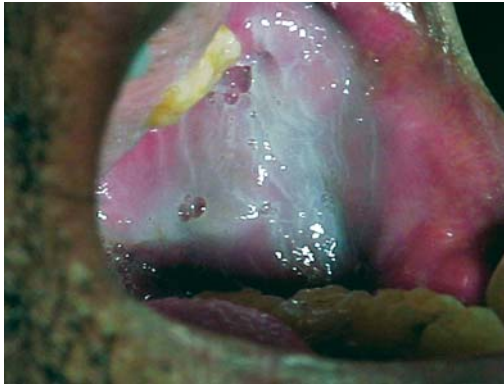


Fig. 9.2: Leukoedema. Note the cloudy white film on the buccal mucosa

represent ectopic sebaceous glands and are of developmental in origin (see chapter 15). Often these lesions are seen as multiple yellowish white lesions of granular appearance. When several lesions coalesce they may be seen as patches.

Frictional keratosis (Fig. 9.3)

Frictional keratosis is a white lesion that is caused by chronic friction which results in the formation of excessive amounts of keratin. Chronic local trauma may be derived from cheek biting, dental appliances, or sharp cusp of tooth impinging on cheek mucosa. Condition is reversible on removal of the cause. History of friction/chronic irritation is suggestive.

Smoker's keratosis (Fig. 9.4)

Smokers Keratosis is a condition caused by chronic tobacco smoking which results in the formation of excessive amounts of keratin.



Fig. 9.3: Frictional keratosis (Arrow) caused by friction derived from sharp buccal cusps of the mandibular left first molar



Fig. 9.4: Smoker's keratosis. White lesion on the hard palate is due to excessive amount of keratin that has resulted from chronic tobacco smoking

Key features include:

- History of smoking
- Asymptomatic in majority of cases
- On the palate (smoker's palate) it is characterized by white palatal mucosa with red nodules representing inflamed orifices of palatal salivary glands
- Condition is reversible on cessation of smoking
- If ulceration or change in the surface thickness of white/red areas occur, biopsy should be taken and lesion should be assessed histologically to rule out pre-malignancy/malignancy
- Smoker's keratosis is not a pre malignant lesion

Chronic hyperplastic candidiasis (candidal leukoplakia) (Fig. 9.5)

This is a white lesion caused by chronic infection by *Candida albicans* which results in hyperplasia of the epithelium.



Fig. 9.5: Chronic hyperplastic candidiasis (candidal leukoplakia). These lesions on the labial commissure are generally bilateral and common in smokers

Key features include:

- White patches often bilateral on angles of the mouth
- Common amongst smokers
- Lesions are triangular in shape with their base towards the labial commissures
- Asymptomatic in most cases
- Very small percentage of lesions may possess malignant potential
- Biopsy is essential to determine malignant potential
- Those patients with no malignant change may respond to anti-fungal topical therapy and cessation of smoking.

Oral hairy leukoplakia (Fig. 9.6)

This is a hairy white lesion seen on the lateral borders of the tongue in HIV disease or those with immune deficiency.



Fig. 9.6: Oral hairy leukoplakia on the lateral border of the tongue in an HIV patient

Key features include:

- Asymptomatic in most cases
- Commonly seen on lateral borders of the tongue in patients with HIV/AIDS disease or those whose immune system is severely compromised (renal transplant patients on immunosuppressive therapy for example)
- White hair-like structures with corrugated appearance on lateral border of tongue are characteristic
- Epstein-Barr Virus is the causative agent
- High doses of antiviral agents (acyclovir) may yield regression of lesions
- Oral Hairy Leukoplakia is not a pre-malignant lesion/condition.

Leukoplakia

The term leukoplakia simply means a white patch. Leukoplakia is a pre-malignant lesion. Not all white

lesions possess malignant potential; hence, the term Leukoplakia has been reserved for a non-scrapable white lesion that cannot be classified as any other known entity (such as lichen planus, candidal leukoplakia, etc.) (Fig. 9.7).



Fig. 9.7: Leukoplakia. Note a thick white patch on the left buccal mucosa. This is a pre-cancerous lesion

Key features include:

- Leukoplakia may be idiopathic
- Tobacco habits and alcohol use may be associated as causative agents
- Lesion is often asymptomatic
- Leukoplakia presents as a white patch adherent to the underlying tissues
- Different clinical appearances such as speckled (red and white), wrinkled, fissured, and verrucous forms may be seen
- Any part of the oral mucosa may be involved, buccal mucosa is the most common site
- 8-10 percent of leukoplakias carry malignant

potential, hence considered as a pre-malignant lesion

- Ulceration of leukoplakia is a disturbing sign which may signify pre malignancy or malignancy
- Histology of the lesion shows dysplastic changes of varying degrees.

Syphilitic leukoplakia

A rarely occurring white patch caused by tertiary syphilis. Also known as syphilitic interstitial glossitis. Syphilitic white patch usually is seen on the dorsum of the tongue. Clinically appears as a white thick patch firmly adherent to the underlying tissues. The condition may carry malignant potential.

Lichen planus

This is a chronic dermatological disorder generally involving skin and oral mucosa (Fig. 9.8).

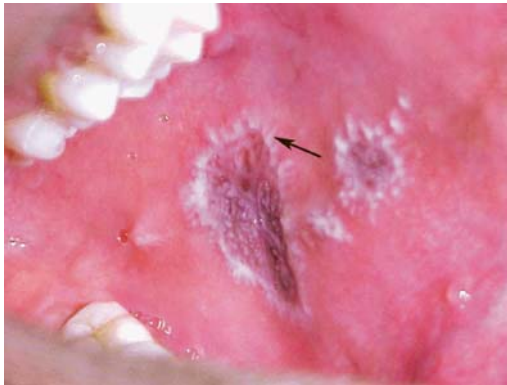


Fig. 9.8: Lichen planus of the right buccal mucosa. Note the lace-like white streaks at the periphery of the lesions (Arrow). This particular example of lichen planus also shows pigmentation

Key features include:

- Often patients present Lichen Planus lesions only on the oral mucosa
- Clinically Lichen Planus is characterised by any of the following appearances: lace-like, plaque-like, papular, atrophic and bullous types
- Common in middle aged women
- Burning mouth may be present
- Usually bi-lateral in distribution
- Stress may be one of the possible causes
- Buccal mucosa, retromolar areas, gingivae, and lips are commonly involved sites
- A small percentage of erosive types are known to carry malignant potential (pre malignant condition)
- Lesions similar to Lichen Planus may occur as a result of drug reactions. These are called Lichenoid reactions
- Corticosteroid topical preparations are used to treat this condition
- Biopsy is warranted for erosive lesions as these are considered to be pre-malignant conditions.

Lupus erythematosus (Figs 9.9A and B)

Two types namely:

1. Systemic lupus erythematosus and
2. Discoid lupus erythematosus are known.

Key Features include:

- This is an immunological disorder
- Butterfly lesions (malar rash) on the face is common in systemic Lupus Erythematosus (Fig. 8.9A)
- Intraoral white patches seen in 20 percent of Lupus Erythematosus patients



Figs 9.9A and B: **A.** Lupus erythematosus. Note the malar rash (butterfly rash) on the face. **B.** Lichen planus-like lesion with white borders (Arrow) on the buccal mucosa

- Intra oral lesions resemble those of Lichen Planus (Fig. 9.9B)
- Very small percentage of Lupus Erythematosus lesions may carry malignant potential.

Squamous cell carcinoma

This is oral cancer originating from epithelial (squamous) cells of oral mucosa. Several clinical forms exist. Squamous cell carcinoma appearing as white patch or white cauliflower like growth is often seen among tobacco users.

Key features include:

- Verrucous carcinoma is a good example (Fig. 9.10A) of a white lesion. Well differentiated squamous cell carcinomas can also present themselves as white patches (Fig. 9.10B)
- Those with heavy tobacco smoking/chewing and alcohol habits are particularly prone to developing squamous cell carcinomas of varying degree of malignancy
- Any firm white lesion that persists for a long time and the one that shows signs of growth and



Figs 9.10A and B: **A.** Verrucous carcinoma and **B.** Squamous cell carcinoma appearing as white lesions. Note the ulceration in the vestibule (Arrow in B) which indicates infiltrating malignancy

ulceration should be biopsied to rule out/or confirm malignant change.

White sponge nevus

This is a genetic (autosomal dominant) disorder involving the oral mucosa as a white patch. Though congenital, often seen only at adolescence. Painless folded white patches are seen on gingivae, buccal mucosa, lips and floor of the mouth (hence also called familial white folded gingivostomatitis). Family history is suggestive.

Dyskeratosis congenita

This is an autosomal dominant congenital disorder characterized by the occurrence of skin and mucosal white lesions. Oral Cavity is involved in 40 percent of the cases. Painless, papular white lesions are seen on the buccal mucosa, palate, tongue and gingivae. Biopsy is confirmatory.

Submucous fibrosis (Fig. 9.11)

This is characterized by a burning sensation of the mucosa following progressive inability to open mouth



Fig. 9.11: Submucous fibrosis. Note the cloudy, opaque appearance of the buccal mucosa. The patient is unable to open his mouth due to intense fibrosis present. This is a pre-cancerous condition

due to the formation of fibrous bands in the sub-mucosa.

Key features include:

- Commonly affects persons of Asian origin—Indians in particular
- Probably caused by chewing betel nut
- Oral mucosa shows opaque white appearance
- Tongue, buccal mucosa, soft palate and lips are involved
- This condition carries malignant potential, hence is considered as a premalignant condition.

Actinic keratosis

This is a white keratotic lesion on the lower lip caused by chronic sun exposure.

Key features include:

- Common amongst outdoor workers

- The lower lip is commonly affected due to the damaging effects of the ultraviolet radiation from chronic sun exposure
- White keratotic flakes seen on vermillion of the lip
- Carries malignant potential.

Suggested reading

1. Prabhu SR. White Lesions of the Oral Mucosa: In Textbook of Oral Medicine. Oxford University Press 2004;91-105.
2. Scully C, Porter S. Orofacial Disease: Update for the Dental Clinical Team 3: White Lesions 1999;123-9.

CHAPTER

10

**Differential
Diagnosis of
the Brown,
Black and Blue
Lesions of the
Oral Mucosa**

SR Prabhu

Learning objectives

After studying this chapter the student should be able to:

- List common pigmented lesions that occur on the oral mucosa
- Understand the etiology of oral pigmented lesions
- Understand clinical features of oral pigmented lesions
- Know what investigations are required to confirm diagnosis of oral pigmented lesions

Lesions with altered coloration are common on the oral mucosa. These are often caused by endogenous or exogenous pigments or deposits of foreign material in the tissues. Malignant melanoma and Kaposi's sarcoma are two examples of colored lesions that require special attention because of their malignant behavior. Recognition of these lesions is important because of the fact that their early diagnosis would lead to a better prognosis. This chapter deals in brief a host of 'pigmented' lesions that clinically present themselves as brown, black or blue lesions.

Clinical presentation of pigmented lesions is variable. They can be seen in any of the following forms:

- Single small round flat spot (macule) of about 1-2 mm in diameter
- Macule of >2 mm in size
- A patch of several centimeters in size and of irregular shape
- Multiple spots
- Multiple patches
- Linear in appearance
- Raised pigmented nodules/patches

Steps to be taken in arriving at a diagnosis of pigmented lesions are:

1. History that includes medical, social and dental history.

2. Intraoral examination.
3. Examination of skin if mucocutaneous conditions are suspected.
4. Clinical photographs.
5. Radiographs where appropriate.
6. Biopsy and histological assessment where necessary.

Classification of brown, black and blue lesions includes:

1. Racial pigmentation
2. Freckle
3. Oral melanotic macule
4. Naevus
5. Smoking associated melanosis
6. Lentigo
7. Melanoma
8. Neuroectodermal tumor of infancy
9. Drug induced pigmentation:
 - Antimalarials
 - Tetracyclines
 - ACTH
 - Zidovudin
 - Clofazimine
 - Busulphan
 - Menthol
 - Contraceptive pill
10. Pigmentation associated with systemic disease:
 - Addison's disease
 - Albright syndrome
 - Multiple neurofibromatosis
 - Peutz-Jegher's syndrome
 - Wilson's disease
 - Gaucher's disease

- Incontinentia pigmenti
- HIV disease
- 11. Amalgam tattoo
- 12. Heavy metal pigmentation
- 13. Tattoos.

Racial (physiological) pigmentation (Fig. 10.1)

This is physiologic pigmentation seen predominantly in dark skinned persons.

Key clinical features

- Band-like diffuse brown-black pigmentation commonly seen at the junction between attached gingival and alveolar mucosa
- Buccal mucosa, palate and tongue are other sites that can be involved.

Differential diagnosis

- Clinically should be distinguished from pigmentation caused by smoking



Fig. 10.1: Racial (physiological) pigmentation

- When present on other mucosal surfaces, drug induced pigmentation and pigmentation of Addison's disease should be ruled out. Medical history is of paramount importance in such circumstances.

Freckles (Ephelis)

Key clinical features

- Common among Caucasians
- Multiple light brown colored flat spots on sun exposed skin
- Spots tend to darken following sun exposure
- In Peutz-Jegher's syndrome spots are multiple and distributed periorally (Fig. 10.2).

Oral melanotic macule

Key clinical features

- Generally a single, large, flat dark brown-black well circumscribed lesion



Fig. 10.2: Note multiple freckles on the upper and lower lips. This is an example of Peutz-Jegher syndrome

- Vermillion aspect of the lower lip is the common site of its occurrence

Smoker's melanosis

Key clinical features

- Smoker's melanosis is due to tobacco habits such as smoking, tobacco chewing or snuff dipping
- Diffuse brown patch commonly seen on anterior gingivae and buccal mucosae
- History of tobacco use is suggestive
- Generally disappears on cessation of tobacco habits.

Differential Diagnosis

- Should be differentiated from racial pigmentation and pigmentation due to medications.

Naevus (mole)

Naevi are hamartomatous lesions made up of focal collections of pigmented naevus cells situated in the connective tissue beneath the epithelium.

Key clinical features

- Common on the skin surface and rarely seen on the oral mucosa
- Presents as a small single painless well circumscribed dome shaped brown-black lesion (Fig. 10.3)
- If ulceration or recent growth is seen in these lesions, biopsy to rule out malignancy should be performed
- Malignant transformation of naevi is possible but rare.



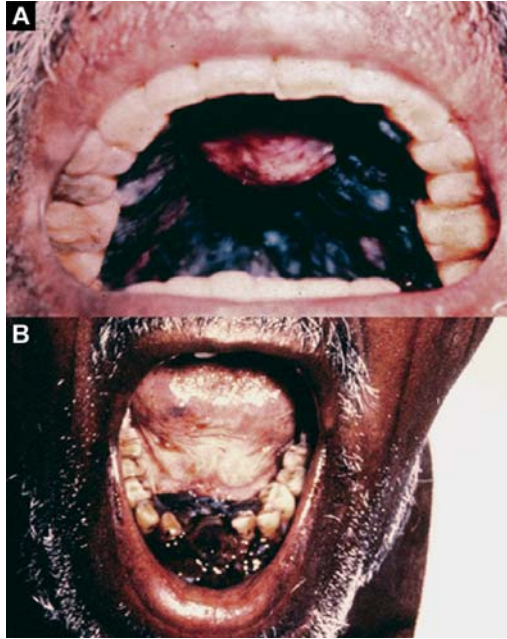
Fig. 10.3: Mucosal naevus on the palatal mucosa

Malignant melanoma

Malignant melanoma arises from pigment producing cells (melanocytes) in the skin and oral mucosa.

Key clinical features

- Oral melanomas are rare
- Oral sites of occurrence include hard palate and gingivae (Figs 10.4A and B)
- Some melanomas arise *de novo* whilst others may arise from pre existing pigmented oral lesions or as metastatic melanomas from other parts of the body
- Melanomas have a poor prognosis
- Three types of melanomas can occur: nodular (invasive), superficial spreading (invasive) and lentiginous (pre-invasive) malignant melanomas
- All forms exhibit dark blue/black lesions with or without evidence of ulceration and inflammation.



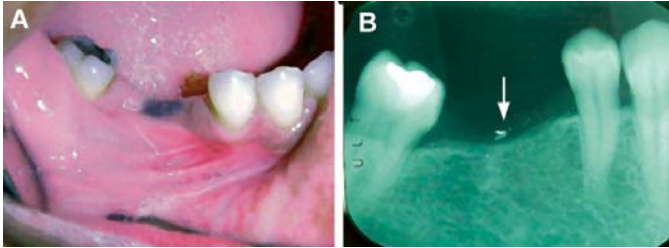
Figs 10.4A and B: **A.** Malignant melanoma involving the palate and **B.** Lower anterior gingivae

Amalgam tattoo

Amalgam tattoo is caused by accidental implantation into the soft tissues of amalgam particles during tooth extraction or replacement of an existing restoration.

Key features include:

- Asymptomatic
- Blue-black non elevated soft tissue lesion
- Alveolar ridge is the most common site (Figs 10.5A and B)
- Large deposits are detectable on X-rays.



Figs 10.5A and B: **A.** Amalgam tattoo. Note the black pigmented lesion on the alveolar mucosa. In the periapical radiograph **B.** Fragments of amalgam can be detected as a radiodense material (Arrow)

Heavy metal pigmentation

Heavy metal pigmentation is caused by excessive ingestion of or exposure to heavy metals such as bismuth, lead, mercury and silver.

Key features include:

- Symptomless in most cases
- Both skin and mucous membranes may be involved
- Lesions range from linear gingival lines to patches
- Metallic taste and gastrointestinal disturbances are often reported
- History of industrial exposure to metals is sometimes positive.

Drug induced pigmentation

Regular use of some medications cause mucosal pigmentation. They include: antiviral agents (*zidovudine* used for HIV), certain antibiotics such as *Tetracycline*, antimalarials such as *chloroquine* and oral contraceptive pills.

The clinical presentation of drug induced mucosal pigmentation is similar to that of heavy metal pigmentation.

Pigmentation associated with systemic conditions

Peutz-Jegher's syndrome

Peutz-Jegher's syndrome is characterized by:

- The presence of multiple nonelevated brown to black spots seen periorally on the skin and around nose and eyes
- Genetic nature (autosomal dominant) and
- Intestinal polyps which may carry malignant potential
- Occurrence in childhood
- Pigmented spots on buccal mucosa and gingivae (see Fig. 10.2).

Addison's disease

This disease is due to idiopathic adrenal cortical insufficiency (Hypoadrenocorticism).

Key features include:

- Hyperpigmentation of the oral mucosa and sun exposed parts of skin as spots or patches
- Buccal mucosa gingivae and tongue may be involved
- Primary adrenocortical insufficiency may be due to autoimmune destruction of adrenal gland
- Secondary adrenal insufficiency is more common which is due to deficient cortisol secretion as a result of exogenous corticosteroid therapy.
- Mucosal pigmentation in secondary adrenal insufficiency is minimal if any

- Patient is lethargic, fatigued, loss of appetite, loss of body hair, irregular menstrual cycle, hypotension, hypoglycemia postural dizziness etc are common.

Albright's disease

This is characterized by fibrous dysplasia of bones and accompanying skin and mucosal hyperpigmentation.

Key features include:

- Deformities of long bones from early childhood
- Pain and aching of involved bones
- Facial bones often involved with slow growing swellings
- Skin and oral mucosa present coffee colored brown spots/patches
- Alkaline Phosphatase levels may be raised
- Histologic diagnosis of bone is necessary.

Neurofibromatosis (von Recklinghausen's disease)

The condition is an heritable disease characterized by the presence of multiple nodular pedunculated lesions on the skin of the trunk, face and arms.

Key features include:

- Nodular lesions on the face, neck and other skin surfaces
- Occasionally diffuse pendulous tissue (elephantiasis neurofibroma)
- Coffee colored patchy pigmentation on the skin and mucosa is generally not involved
- Nodular lesions may be seen intraorally
- A small percentage of these lesions carry malignant potential.

Melanotic neuroectodermal tumor of infancy (Fig. 10.6)

This tumor is rare. Seen in infants, this tumor grows fast in the jaws and is accompanied by pigmented patches in the tumor. Pigment is derived from cells containing melanin.



Fig. 10.6: Neuroectodermal tumor of infancy involving the maxilla. Note the pigmentation on the surface of the tumor (Arrows)

Incontinentia pigmenti

This is a genetically derived dermatosis almost exclusively occurring in females.

Key features include:

- Seen shortly after birth
- Recurrent erythematous vesiculobullous lesions on the skin
- White keratotic lesions and brown patches on the skin at later stages.

Pregnancy

Pigmentation of the skin and occasionally that of the oral mucosa is not uncommon in pregnancy. This is due to stimulation of melanin pigmentation as a result of hormonal changes in pregnancy. Pigmentation is usually of patchy light brown in nature.

Suggested reading

1. Scully C. Slide Interpretation in Oral Disease. Oxford University Press, Oxford 1999;162-77.
2. Wilson DF, Moore SR, Logan RM. Pigmented Lesions of the Oral Mucosa: In Prabhu SR: Textbook of Oral Medicine. Oxford University Press 2004;107-16.

CHAPTER

11

**Differential
Diagnosis of
Red Lesions
of the Oral
Mucosa**

SR Prabhu, H Al Bayaty

Learning objectives

After studying this chapter the students should be able to:

- Classify red lesions of the oral mucosa
- Discuss etiology of different types of red lesions
- Describe clinical features of the more common types of oral red lesions
- Discuss investigative methods employed in those red lesions, which are not easily diagnosed clinically
- Discuss etiology, clinical features diagnosis and therapeutic management of those red lesions, which may possess malignant potential.

Introduction

Oral soft tissue lesions, which clinically appear red, are common. These lesions may present themselves in different clinical forms. They may be patchy, spotted, macular, papular, nodular, localized, diffuse, single multiple and so on.

Examiner should gather information with regard to the possible causes. Lesions that appear red may be primary or secondary as far as their origin is concerned. When they appear as a result of local causes. Sometimes red lesions may be associated with pigmented or white lesions.

- Loss of covering epithelium may be responsible for the redness. Increased capillary blood supply underneath the epithelium, or increased proliferation of vascular tissue may also be responsible for the redness in a given lesion
- Inflammation is the major cause of majority of red lesions. Neoplastic/pre-neoplastic changes in the tissues may also occasionally give rise to red lesions. Examiner should therefore look for cardinal signs of inflammation such as pain,

swelling, increased temperature, etc. Neoplastic or pre-neoplastic red lesions on the other hand may often be asymptomatic unless secondarily infected or ulcerated.

Classification of the red lesions of the oral mucosa

Mucosal red lesions of inflammatory origin

Red lesions due to:

Traumatic causes

- Mechanical irritation
- Chemical injury
- Thermal injury
- Radiation injury.

Red lesions due to infections

- Pharyngitis/tonsillitis
- Scarlet fever
- Chronic erythematous candidiasis
- Infectious mononucleosis.

Red lesions due to immunologic reactions

- Allergic mucositis
- Lupus erythematosus
- Sjögren's syndrome
- Erosive lichen planus
- Erythema multiforme
- Desquamative gingivitis.

Red lesions of non-inflammatory origin

Red lesions of hematologic/vascular causes

- Purpura due to thrombocytopenia

- Ecchymosis due to clotting factor deficiencies
- Hereditary hemorrhagic telangiectasia due to heritable vascular causes
- Hematoma due to extravasation of blood
- Port wine nevus (macular hemangioma) due to developmental defects in the vasculature.

Red lesions due to unknown causes

- Erythroplakia
- Geographic tongue
- Erythema migrans

Red lesions due to nutritional disorders

- Vitamin deficiency glossitis/mucositis
- Iron deficiency glossitis/mucositis.

Red lesions of traumatic origin

Inflammatory changes in the tissues cause redness. Trauma is one of the major factors in this context.

Types of traumatic agents, which can cause redness of the mucosa include any of the following:

Physical/mechanical trauma

- Ill fitting dentures
- Ill fitting orthodontic appliances
- Sharp cusps of teeth
- Chronic habitual irritation.

Thermal trauma

- Pizza burn

Chemical trauma

- Aspirin burn

Trauma from ionizing radiation

- Radiation mucositis

Examiner should look for the source of irritation or trauma.

Pizza burn and aspirin burn are examples of thermal trauma and may be accompanied by white slough. The redness is localized.

Radiation mucositis is characterized by the diffuse redness of the oral mucosa. In these patients there is a history of receiving radiation for cancer.

Management of lesions of traumatic origin includes:

- Elimination of the cause such as ill fitting denture or appliance
- Symptomatic treatment of pain by the use of topical application of local anesthetic gel before meals.

Red lesions due to infections**Pharyngitis/tonsillitis**

Infections of the pharynx, tonsils and larynx often cause redness of the respective sites.

Pharyngitis due to Coxsackie virus Type A for example manifests as multifocal nodules with erythematous base extending on the soft palate and oropharynx. The patient complains of sore throat and general malaise and is febrile. The condition is self limiting and resolves in about a week.

- The cause of bacterial pharyngitis is pyogenic streptococcus. Bacterial pharyngitis is often accompanied by bacterial tonsillitis (pharyngo-tonsillitis) in which, pharyngeal mucosa is bright red, uvula is edematous and soft palate is inflamed

and red. Swollen tonsils with exudates in their crypts may often be seen. Cervical lymph nodes are palpable and tender. Bacterial pharyngitis responds to antibiotics. Culture studies are to be obtained and antibiotics started empirically

- Rapid antigen detection tests for group A streptococci have been useful with about 80 percent sensitivity and 90 percent specificity
- Proper and prompt treatment of streptococcal pharyngitis lessens the possibility of rheumatic heart disease and post streptococcal glomerulonephritis
- Analgesics, saline gargles and plenty of fluids are the essential steps towards the supportive treatment.

Scarlet fever

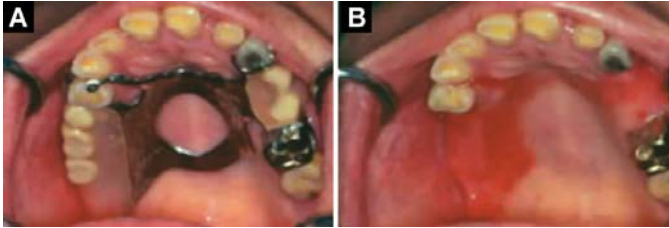
In people with streptococcal pharyngitis scarlet fever is common. In these patients Erythrogenic toxin elaborated by streptococcal organisms causes the damage.

- Patient's tongue shows protruding papillae (strawberry tongue) or a bright red tongue with large papillae (raspberry tongue)
- Scarlet fever also manifests erythematous skin rash, cervical lymphadenopathy, sore throat, headache, vomiting and fever.

A culture is diagnostic. Intravenously administered penicillin is the treatment of choice.

Chronic erythematous candidiasis

This condition is common among denture wearers (Figs 11.1A and B) although non denture wearers



Figs 11.1A and B: **A.** Chronic erythematous candidiasis of the palate in a partial denture wearer. Note the partial denture in place. **B.** The redness of the denture bearing mucosal surface

may also show this condition. In denture wearers this is commonly known as denture sore mouth. There is no denture allergy involved in this condition.

- The agent responsible for erythematous candidiasis is *Candida albicans*. Diffuse red areas on the denture bearing surfaces of the palate are the hall marks of this condition
- Often angular cheilitis characterized by cracked corners of the lips is associated with this condition. On the palate occasionally white curd-like lesions that are easily removable by digital pressure are also seen
- Treatment of denture sore mouth includes application of antifungal (nystatin) creams to the denture base followed by relining of the denture. Erythematous candidiasis not related to the denture is treated with nystatin oral suspension. Examples of erythematous candidiasis among non-denture wearers includes long term use of antibiotics or topical steroids.

Infectious mononucleosis

Infectious mononucleosis (Glandular fever) is caused by Epstein-Barr virus. The disease is common among

young adults and is clinically characterized by malaise, fever, upper respiratory tract infection, cervical lymph node enlargement and hepatosplenomegaly.

- Oral findings of infectious mononucleosis are characterized by pin pointed petechial red spots on the soft palate. Diffuse inflammation of the mouth may also be present. Epistaxis is evident in some patients
- Condition is self limiting. Management includes fluid intake and bed rest
- Symptomatic treatment for fever and pain is recommended in most cases.

Red lesions due to immunologic reactions

Allergic stomatitis (Allergic mucositis)

Immediate hypersensitivity to drugs or contact allergy may often lead to inflammatory oral mucosal red lesions. Antibiotics, sulfonamides, barbiturates and iodine are capable of causing hypersensitive reactions.

- Diffuse mucosal redness is the hall mark of allergic mucositis. Pseudomembrane and desquamation of epithelium may occur in some cases.

Contact allergic response on the other hand is generally discrete. The redness occurs in the area that is in direct contact with the allergen.

- Some known allergens include toothpaste, dental materials, topical medications, cosmetics and so on. Allergen in the chewing gum can sometimes evoke a generalized erythematous response of the oral mucosa and the gingiva. This condition is called plasmacytosis gingivae

- Burning of the mucosa or itchiness are consistent features in both hypersensitive and contact allergy patients.

History and elimination of the known allergen are of utmost importance in the diagnosis and management of these conditions. Antihistaminics help to control systemic reactions and Triamcinalone acetone in orabase is useful for contact allergy.

Lupus erythematosus

Lupus erythematosus (LE) is an autoimmune disease. Two types of the disorder exist: Systemic Lupus Erythematosus (SLE) and Discoid Lupus Erythematosus (DLE).

- Oral lesions in LE include a combination of red and white lesions (Fig. 11.2B). They are often indistinguishable from Lichen planus
- Buccal mucosa with diffuse involvement is commonly seen
- A typical “butterfly rash” over the nasal bridge and malar eminence bilaterally (Fig. 11.2A) is typical of SLE
- Clinical diagnosis of LE is based on the skin involvement. Detection of antinuclear antibody (ANA) and LE preparation are helpful in confirmation of the disease. Lupus erythematosus is treated by systemic immunosuppressant therapy.

Sjögren’s syndrome

Sjögren’s syndrome (SS) is an autoimmune disorder, which mainly affects the salivary glands, lacrimal apparatus and occasionally the joints.



Figs 11.2A and B: **A.** Lupus erythematosus. Note the bilateral “butterfly” redness on the cheek bones **B.** Intraoral lesions of lupus erythematosus appear identical to those of lichen planus

- Dryness of the mouth is a prominent feature of this disorder
- Atrophy and the associated redness of the mucosa is often seen, particularly on the tongue
- The diagnosis and treatment of the disorder is largely the responsibility of the physician. Xerostomia can be managed by the use of saliva substitute. In severe cases of dry mouth, the use of sialogogue such as pilocarpine HCL may offer some relief
- Caries control by appropriate topical fluoride applications is necessary in these patients.

Erosive lichen planus

- Erosive form of the disease is one of the three clinical types of lichen planus and is characterized by red patches with white radiating streaks at its borders (Fig. 11.3)
- Red lesions which are generally bilateral in distribution are found frequently on the buccal mucosae and the mucobuccal fold of the mouth
- Occasionally, erosive form of the disease is preceded by bullous lesions



Fig. 11.3: Erosive lichen planus of the buccal mucosa

- Lesions are painful and mucosal burning is common. Patients often complain of insensitivity to hot drinks and spices used in the food
- It is to be noted that a small proportion of these lesions tends to possess malignant potential and are therefore considered as premalignant conditions
- Diagnosis of erosive lichen planus is relatively easy on clinical grounds alone. Histopathologic examination of the biopsied material is confirmatory
- Treatment includes topical application of steroids. In severe cases systemic use of steroids may become necessary. Periodic follow-up of the patient is important.

Erythema multiforme (EM)

Erythema multiforme is a condition that may be precipitated by parenterally administered medications or viral infections.

- This mucocutaneous disorder exhibits characteristic “iris lesions” on the skin and diffuse red lesions on the oral mucosa

- Often blisters and ulceration may accompany oral red lesions. Any part of oral mucosa may be involved showing a combination of redness and erosion in EM (Fig. 11.4A)
- A more severe form of the disease called Stevens-Johnson syndrome is characterized by oral (Fig. 11.4B), conjunctival and genital mucosal involvement
- History of drug use or viral infection is suggestive in most cases. Discontinuation of the medication and low-doses of systemic steroids are of help
- Topical application of local anaesthetic agents or antihistaminic elixir rinse are helpful in the elimination of mucosal pain.

Desquamative gingivitis

Desquamative gingivitis is not a specific disease entity. It is a clinical sign of a disease such as lichen planus or pemphigus involving the gums.

Clinically desquamative gingivitis is diffusely erythematous (Figs 11.5A and B). The condition is painful. Treatment generally includes the use of topical steroids and topical anaesthetic agents.



Figs 11.4A and B: **A.** Erythema multiforme and **B.** Stevens-Johnson syndrome. The latter is a severe form of erythema multiforme



Figs 11.5A and B: Examples of desquamative gingivitis. The gingival epithelium is denuded giving rise to extensive erosion

Red lesions due to non-inflammatory causes

Red lesions due to haematologic/vascular defects

Purpura in thrombocytopenia

- Muco-cutaneous manifestations of thrombocytopenia include petechiae
- Red pin-pointed lesions (Fig. 11.6) are the hall marks of this condition. These are also accompanied by gingival haemorrhage
- Post extraction bleeding will be prolonged in thrombocytopenia
- The condition is characterized by the deficiency of circulating platelets. This deficiency can result from primary thrombocytopenia or platelet defect arising from bone marrow depression
- Clinically the torniquet test is positive in thrombocytopenia.

Diagnosis rests on a complete blood count, and platelet count. History often suggests the use of drugs, which may have caused bone marrow depression leading to thrombocytopenia.

Management of thrombocytopenia is the responsibility of a hematologist.



Fig. 11.6: Purpuric spots of thrombocytopenic purpura on the palate

Ecchymosis due to clotting factor deficiencies

Ecchymosis is a feature resulting from extravassation of blood. Extravassation of blood with no known traumatic episode should raise suspicion of defective clotting mechanism. This differs from petechiae only in size.

Ecchymosis occurs in systemic conditions such as:

- Hemophilia and Christmas disease
- Anticoagulant therapy
- Primary liver disease.
- The submucosal redness in these patients is often seen by the treating dentist for the first time
- The lesions may be slightly raised due to the submucosal collection of the extravassated blood
- The color may show variation in color range depending on the length of time the extravassated (unclotted) blood has been present under the

mucosa. Oral lesions may often be seen as blisters filled with blood.

Diagnosis rests mainly on finding out the defect in coagulation factors. Tests such as partial prothrombin time (PT), bleeding and clotting time (BT and CT), and factor assays are to be carried out. Extractions or oral surgical procedures should not be carried out in these patients until the defect has been corrected.

Hereditary hemorrhagic telangiectasia

Hereditary telangiectasia is an inherited disorder characterized by the appearance of red spider telangiectasiae of the skin and petechiae-like lesions of the oral mucosa (Fig. 11.7).

Perivascular supportive tissue seems to be weak causing petechial lesions. Bleeding from the nose and gums is often reported.

- Family history and clinical appearance of the lesions are suggestive



Fig. 11.7: Hereditary haemorrhagic telangiectasia. Note the raised pin-pointed red vascular lesions on the tongue

- Bleeding and clotting time tests and tests for platelets and clotting mechanism are normal in these patients
- Oral bleeding can generally be controlled by pressure packs.

Hematoma

Hematoma results from the extravasation of blood in a confined space due to trauma. These lesions clinically resemble hemangiomas but when compressed do not blanch. These mimic ecchymoses as well.

- There is a history of trauma
- Lesions may be red to brown or black depending on the duration of the extravasated (clotted or partly clotted) blood. Fresh lesions are red in color. Lesions may often be raised
- Hematomas resolve in about 2 weeks. Since melanoma, Kaposi's sarcoma, hemangiomas and hematoma have similar clinical appearances; a careful follow up is required particularly when there is no history of trauma in cases of suspected hematoma.

Port-wine nevus

Vascular malformations resulting in red lesions of the skin and mucosa are called vascular nevi.

- The vascular defects seen on the face along the distribution of the trigeminal nerve are called nevus flammeus. This red (port wine in color) vascular malformation is unilateral and does not cross the midline (Fig. 11.8).

When traumatized this lesion bleeds profusely.



Fig. 11.8: Port-wine nevus of the palate. Note the redness that does not cross the midline

Because of the vascular malformation, lesions of this type elicit blanching on pressure. Often seizures (due to intracranial calcifications) are reported in these patients. This condition is also called Sturge-Weber syndrome.

Red lesions due to unknown causes

Erythroplakia

Erythroplakia, literally means a red patch. Traditionally this term has been reserved for a condition that possesses malignant potential in a significant proportion of patients.

- The condition is bright red (velvety) in appearance with well-defined sharp borders (Fig. 11.9)
- The surface of the lesion is slightly raised and pain is not a feature of this condition hence patients may be unaware of its presence in the mouth
- Etiology of this condition is not known. A



Fig. 11.9: Erythroplakia of the ventro-lateral border of the tongue

significant proportion of patients however smoke tobacco or abuse alcoholic

- Oral floor, soft palate buccal mucosa and ventral surface of the tongue are frequently involved sites.

As a rule of thumb, any red lesion that cannot be identified as any other known entity and that has been persistently present for few weeks/months despite the routine treatment should be biopsied. In erythroplakia dysplastic changes in the spinous and basal cell layer are evident suggesting pre-malignant nature of the lesion.

Treatment includes wide local excision of the lesion and follow-up.

Geographic tongue

Geographic tongue, also known as migratory glossitis is a common condition of the tongue often seen in childhood or early adult life.

- The dorsum of the tongue and occasionally the lateral borders of the tongue are sites of this condition
- Red islands occur as a result of loss of filiform papillae. The fungiform papillae are inflamed and mildly swollen
- The red map-like islands are surrounded by white keratotic rims giving a characteristic map-like appearance
- These structures tend to wax and wane and appear in different locations during recurrence (migratory glossitis). Patients are generally unaware of these lesions. Occasionally, however they may complain of sore tongue
- This condition does not need any treatment. Those patients with cancer phobia should receive reassurance as to its benign nature. If burning sensation is present, appropriate symptomatic treatment can be given.

Erythema migrans

Erythema migrans is similar in clinical presentation to geographic tongue. The major difference however is that the lesions appear on soft palate, buccal mucosa and ventral tongue mucosa.

This condition is not common. As for geographic tongue erythema migrans does not require any treatment. Reassurance is adequate.

Red lesions due to nutritional deficiencies

Glossitis due to vitamin deficiencies

Vitamin deficiencies are common in certain communities and countries due to nutritional

deficiencies. In the affluent part of the world, vitamin deficiencies can occur mainly among the alcoholics.

- Deficiency of riboflavin, niacin and cyanocobalamin can result in pernicious anemia. Folic acid deficiency and sprue commonly cause atrophy of the lingual mucosa giving rise to smooth and diffuse redness. Often patients complain of burning sensation of the tongue
- Hematologic examination will lead to the diagnosis. Often gastric analysis and bone marrow aspiration may be necessary as confirmatory tests
- Proper diet and vitamin supplement usually corrects the situation
- For non-tropical sprue gluten-free diet should be recommended
- Pernicious anemia can be treated by vitamin B₁₂ injections.

Glossitis due to iron deficiency anemia

Iron deficiency anemia is a common condition in many parts of the world.

- Oral changes in iron-deficiency include atrophic mucosa, which often gives rise to bald tongue and diffuse redness. Patients often complain of burning sensation of the tongue. There are also other general symptoms such as weakness, shortness of breath and tingling sensation in the extremities. Hemogram and estimation of iron levels (ferritin) in blood are essential.

Treatment includes iron supplementation.

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CHAPTER

12

**Differential
Diagnosis of
Ulcers of the
Oral Mucosa**

SR Prabhu, H Al Bayaty

Learning objectives

After studying this chapter the student should be able to:

- List different kinds of ulcers that occur in the oral mucosa
- Understand clinical features of different types of ulcers of the oral mucosa
- Understand the rationale for special investigations that may be necessary in some types of ulcers of the oral mucosa.

Although ulcerations of the oral mucosa are commonly seen in dental practice, majority of these heal within a week or two with or without treatment. In most instances their cause can be traced through history. A small percentage of ulcers however do not heal despite routine treatment or elimination of the cause. These ulcerative lesions must be viewed with high degree of suspicion because of the possibility that these may carry malignant potential or be frankly malignant at the time of diagnosis. Some of these may even be examples of secondary involvement of chronic specific infections such as pulmonary tuberculosis. Histological diagnosis is therefore important in such cases.

Causes of oral ulcerations are multiple. These may operate at local or systemic levels. History, thorough clinical examination and appropriate investigations must be carried out to arrive at a diagnosis of ulcerative lesions.

Clinician should include the following in history taking exercise:

- Onset
- Duration and causes if known
- Recurrence (if any)
- Progression

- Developments that precede the appearance of ulcer(s) (such as formation of vesicles prior to ulceration)
- Appearance of ulcers on other regions of the body (e.g. skin, eye, genitalia)
- Associated symptoms such as fever, malaise, enlargement of the lymph nodes, etc.
- Current medication.
- Overall health status of the patient.

During the clinical examination of ulcers important aspects to be noted are:

- Anatomic location
- Number
- Size
- Shape
- Color
- Margins
- Base
- Edges
- Texture
- Tenderness
- Presence of discharge
- Presence of slough
- Lymph node involvement
- Involvement of other systems such as skin conjunctiva, etc.

Ulcers involving the mucosa are briefly discussed below:

Recurrent aphthous ulcers (RAU)

Recurrent oral ulcers are common. These are painful recurrent ulcers of uncertain etiology. Recurrent ulcers are exclusively seen on the non-keratinized

oral mucosa. Three forms of RAUs are: Minor, Major and Herpetiform aphthous ulcers.

Recurrent minor aphthous ulcer

- Most common form of recurrent oral ulceration (85%)
- *Cause:* Not known; often stress is linked with their occurrence
- *Size:* Usually 2-3 mm in diameter
- *Number:* Single or in groups on the oral mucosa
- *Duration:* ranges from 3 days to 2 weeks
- *Shape/color:* Round/oval with red halo with grey/yellow base (Fig. 12.1).
- *Location:* Nonkeratinised oral mucosa such as labial mucosa
- *Symptom:* painful.

Recurrent major aphthous ulcer (Fig. 12.2)

- *Size:* Greater than 1 cm in diameter
- *Outline:* Irregular



Fig. 12.1: Minor aphthous ulcer on the labial mucosa



Fig. 12.2: Major aphthous ulcer involving the soft palate

- *Number:* Usually single
- *Location:* Non keratinized mucosa in majority of cases such as soft palate/faucus
- *Duration:* May last for weeks to months
- Scar formation can occur on healing
- *Symptoms:* Painful ulcers and can bleed more easily.

Herpetiform aphthous ulcer

- Clinically these ulcers appear similar to ulcers of herpetic stomatitis
- *Size:* Upto 2 mm in size
- *Number:* Can occur in groups of up to 100 or 200
- *Duration:* last a few days to 2 weeks
- *Location:* non-keratinized oral mucosa
- *Symptoms:* Ulcers are painful.

Traumatic ulcer

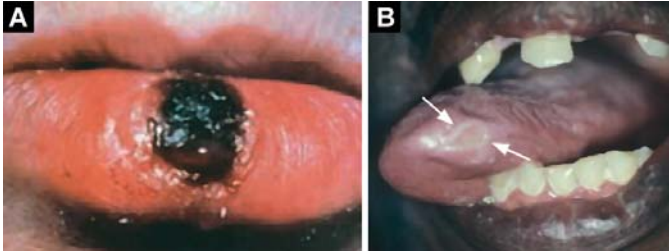
- *Causes:* Trauma arising from sharp edge of fractured tooth cusp or oral appliance, thermal burn, or aspirin burn, patient reports a history of trauma

- *Number:* Usually single
 - *Size/shape:* Variable in size and shape, round/irregular
 - *Margin and the base:* Red margin and yellowish base
 - *Behavior:* Heals within a few days once the cause is removed
- Note:** Any ulcer that does not heal after 2 weeks even after removal of the cause should be biopsied and studied histologically to rule out malignant growth.

Syphilitic ulcer

Syphilitic ulcers may be seen in three stages of the disease, namely; primary, secondary and tertiary syphilis.

- The ulcer of primary syphilis is called a *chancre* (Fig. 12.3A).
- *Causative organism:* *Treponema pallidum*, a spirochete
- *Size:* Chancre can be 0.5 mm to several centimeters in its greatest dimension
- *Shape and edges:* Chancre is a round ulcer with raised/indurated edge
- *Number:* usually single
- *Behavior:* Usually heals spontaneously
- *Symptoms:* Ulcers of secondary syphilis are multiple and painless
- Secondary lesions are flat, irregular and covered by grey membrane which coalesce to form *mucous patches* (Fig. 12.3B)
- The painless ulcer of tertiary syphilis is known as *gumma*
- The *gumma* affects the palate and a few mm to several cm in diameter



Figs 12.3A and B: **A.** Primary syphilitic ulcer of the lower lip and **B.** Ulcer of the tongue (arrows) in secondary syphilis

- Gummatous ulcer has a 'punched out' appearance and the base of the ulcer is pale
- Dark field illumination microscopy and serological investigations are used to confirm syphilis.

Ulcers due to primary herpetic gingivostomatitis

- Primary gingivostomatitis is caused by herpes simplex virus (HSV) Type 1 in 90 percent of cases and HSV 2 in about 10 percent of cases
- Condition is common in children. Rarely adults are infected
- Relatively common in immunodeficient (HIV infection) persons as shown in Fig. 12.4
- Multiple vesicles appear first which rupture and form erosions or ulcers. These ulcers often resemble those of cyclic neutropenia
- These oral ulcers are painful
- The lips become crusty
- Symptoms include fever, nausea, vomiting and headache
- *Shape and size:* Ulcer is round and 2-3 mm in diameter with yellowish floor with a red inflamed boundary



Fig. 12.4: Multiple ulcers due to herpes simplex virus infection of oral mucosa in an HIV infected adult male

- The vesicles can be seen to coalesce at the vermillion border/skin junction; generally seen on keratinized mucosa
- The active phase of the disease will allow detection of antibodies to this virus.

Ulcers due to recurrent herpes infection

- Caused by reactivation of herpes simplex virus
- Vesicles appear on the lips/mucocutaneous junction (herpes labialis/‘cold sore’) which rupture leaving erosions/ulcers
- Common in adults
- Tingling sensation is often felt prior to the formation of vesicles (prodromal symptoms).

Ulcer due to histoplasmosis

- The cause of this disease is a fungus called *Histoplasma capsulatum*
- Common in immunocompromised patients
- The ulcers can occur any where in the mouth and are multiple

- The ulcer is round with the floor covered with a greyish membrane
- Biopsy is required for a definitive diagnosis.

Ulcer due to mucormycosis

- This disease is caused by fungi of the family *Mucoraceae*
- This disease commonly occurs in immunocompromised individuals in the tropics
- Oral ulcers can sometimes be seen
- Culture and biopsy are used for diagnosis.

Ulcer due to cryptococcosis

- This disease is caused by a fungus called *Cryptococcus neoformans*
- This disease occurs in individuals who are immunocompromised
- The ulcers are painful and can occur as single or multiple ulcers
- The palate is affected
- Ulcer upto several cm in size may be seen in the oral cavity
- Biopsy of the affected area and a culture is needed for definitive diagnosis.

Ulcer due to blastomycosis

- This disease is caused by a fungus called *Blastomyces dermatitidis*
- It occurs predominantly in men
- Nodules and ulcers occur in this disorder occasionally involving oral mucosa
- Ulcers occur in multiple numbers
- Differential diagnosis includes ulcers caused by other fungi

- Biopsy and histopathology examination are needed for diagnosis.

Squamous cell carcinoma (SCC) appearing as an ulcer

- SCC is a common malignant condition of the oral cavity
- Occurs mostly in men above 50 years of age
- The use of tobacco products and alcohol are the primary cause
- Presents as a painless long standing non-healing ulcer (Fig. 12.5)
- Commonly lateral border of tongue is involved but can also occur anywhere on the oral mucosa
- Ulcer is indurated and fixed to underlying tissues
- The floor of the ulcer has a granular surface and is fixed to underlying tissues
- Edge of the ulcer is rolled, raised, and everted
- Shape of the ulcer is usually round but can also be irregular



Fig. 12.5: Squamous cell carcinoma of the posterolateral border of the tongue (arrow) appearing as an ulcer

- Differential diagnosis includes all non-healing ulcers such as those due to tuberculosis, syphilis, sialometaplasia of minor salivary glands, etc.
- Biopsy and histopathological examination are mandatory for the diagnosis.

Ulcerated Kaposi's sarcoma

- A connective tissue malignancy increasingly (though not exclusively) seen in HIV infected patients
- Human Herpes Virus (HHV) 8 is the causative agent
- The virus affects the endothelium of the capillaries and causes malignancy
- Single multiple bluish red occasionally ulcerated ulcers on the palate are typical of the disease
- The floor of the ulcer is covered by a grayish-yellow necrotic pseudomembrane
- The ulcer diameter is from a few millimeters to several centimeters
- Small ulcers can coalesce and form larger lesions.
- Differential diagnosis includes hemangioma, malignant melanoma and other non-healing ulcerative lesions
- Biopsy and Histopathology examination are essential for diagnosis
- HIV test should be carried out.

Ulcerated non-Hodgkins lymphoma

- This is a malignant tumor of lymphoid tissue which might lead to ulceration
- Ulcers on oral mucosa are round/irregular
- Floor of the ulcer is yellowish and the margin is erythematous
- Differential diagnosis includes all non-healing ulcers

- Biopsy and histopathology examination are essential for diagnosis of the disease.

Ulcerated malignant melanoma

- This is a malignancy arising from the melanocytes in the basal layer of the surface epithelium
- It is rarely seen in the mouth as an ulcerated pigmented lesion
- Lesion is a few mm to about 1 cm in diameter with an irregular outline
- Melanoma is dark brown/blue in color and may be ulcerated at the time of diagnosis
- Metastasis to cervical lymph nodes is common in this condition
- Prognosis is usually poor
- Differential diagnosis includes ulcerated naevi, Kaposi's sarcoma and pigmented lesions of endogenous origin
- Biopsy is confirmatory for diagnosis.

Ulcers due to malignant tumours of the minor salivary glands

- May present as an ulcerated swelling in the oral cavity, typically on the palate (e.g. adenocarcinoma)
- Ulcer is painful and maybe several centimeters in diameter
- Biopsy and histopathological examination are mandatory for diagnosis
- Differential diagnosis includes all non-healing ulcers including necrotizing sialometaplasia.

Ulcers due to mucous membrane pemphigoid

- This is an autoimmune disease causing loss of

epithelial attachment to the underlying connective tissue

- This condition occurs in patients above 50 yrs of age
- Vesicles and bullae rupture to form ulcers on oral mucosa
- The gingiva and soft palate are particularly affected
- Size of the ulcer can be up to several centimeters in diameter
- Shape of the ulcer is round/irregular
- Edge of the ulcer has a well defined margin
- Ruptured bullae have an inflamed base
- Often involves the conjunctiva resulting in scar formation and blindness
- Biopsy and immunofluorescent microscopy (direct and indirect) are needed for diagnosis.

Ulcers due to pemphigus vulgaris

- This is an autoimmune disease of skin and mucous membrane with the formation of intraepithelial vesicles and bullae
- More common in women 40-60 yrs of age
- 50 per cent of cases initially present with oral lesions
- Painful oral ulcers may result as a rupture of vesicles or bullae
- Buccal mucosa, palate, gingival are affected
- Multiple ulcers occur
- Ulcers may be few millimeters to several centimeters in diameter
- Shape may be irregular with a ragged border
- Floor of the ulcer is shallow with denuded base and is covered with white or blood stained exudates

- Diagnostic tests includes stroking the mucosa which produces bulla or vesicle—this is Nikolsky's sign
- Biopsy shows *acantholysis*
- Immunofluorescent antibody test demonstrates binding of IgG, IgM, and C3 deposits in the intercellular substance of the epithelial tissue and a raised titer of IgG respectively.

Ulcers due to erosive lichen planus

- Although exact cause is not known. This condition has an immunologic background
- Patients present with both skin and lesions of the oral mucosa
- Ulcerative or erosive lesions can occur in the mouth (Fig. 12.6)
- Ulcers can occur anywhere in the mouth
- The ulcer is shallow and irregular in shape



Fig. 12.6: An erosion/ulcer in the vestibule due to erosive lichen planus

- The ulcer may be up to a few centimeters in size
- The ulcer floor is yellow and the margin is red
- Biopsy is needed for definitive diagnosis.

Ulcers due to necrotizing sialometaplasia

- This disorder affects the minor salivary glands of the palate
- The cause is unknown, but often may be due to local ischemia caused by local anesthetic injections
- Ulcers can occur in the affected area
- The ulcer occurs on the palate and is painful
- The ulcer can be up to 2 cm in diameter
- The ulcer is round in shape
- The floor is yellowish
- The edge of the ulcer is indurated (Fig. 12.7).



Fig. 12.7: Sialometaplasia of the minor salivary glands on the hard palate

Ulcers due to tuberculosis

- This disease is caused by bacteria called *Mycobacterium tuberculosis*
- In this disease ulcers can occur in the mouth although not common
- The ulcer occurs on the dorsal/lateral surface of the tongue
- Ulcer is yellowish white at its floor
- Edge of the ulcer is undermined
- Shape of the ulcer is irregular
- Investigations for TB are essential
- Ulcer should be biopsied and tissue processed using Acid Fast Staining (Ziehl-Neelsen) techniques.

Ulcers due to cytomegalovirus (CMV)

- This virus causes salivary gland swelling
- This disease is common in immunocompromised individuals
- Oral ulcers occurring in this condition are painful.
- The tongue and buccal mucosa are commonly affected
- Grayish base of the ulcer is a feature
- Up to 1 cm in diameter is the dimension of the ulcer
- Undermined edges are a feature of the ulcers
- Ulcer may be above 1 cm in size and irregular

Ulcers due to herpangina

- This disease is caused by Coxsackievirus
- Dysphagia, loss of appetite, and fever are observed
- Ulcers up to 2 mm can occur on soft palate
- Border of the ulcer is erythematous
- Gingivae are spared. This is a differentiating point to rule out herpetic gingivostomatitis

Ulcers due to radiation damage

- Often seen in a head and neck cancer patient receiving radiation therapy
- Ulcers may be irregular in shape
- They are extremely painful
- Treatment includes the use of analgesics and mouthwashes. Topical local anaesthetic agents are also useful in relieving pain
- In these patients salivary flow is reduced
- Patients should be given saliva replacement therapy such as artificial saliva.

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CHAPTER

13

**Differential
Diagnosis of
Intraoral
Blisters**

SR Prabhu, H Al Bayaty

Learning objectives

After studying this chapter the student should be able to:

- Understand general features of vesicles and bullous lesions that occur in the mouth
- List all blister forming conditions of the oral mucosa
- Understand the etiology of blister forming conditions of the oral mucosa
- Know diagnostic aspects involved in blister forming conditions.

**Blister forming diseases:
general features**

Blisters are fluid filled lesions of different sizes. Those which are less than a centimeter in diameter are called vesicles. Blisters greater than one centimeter are considered as bullae. In some conditions vesicles may coalesce to form bullae. Collectively these lesions are known as vesiculobullous lesions. Vesiculobullous lesions may sometimes involve both oral mucosa and the skin.

- Most vesicular diseases are of viral origin
- Majority of bullous diseases have an immunological background
- Some bullous diseases may have serious consequences in the absence of treatment
- Most bullous diseases involve both skin and mucous membrane
- Often in vesicular disease multiple vesicles coalesce to form bullae
- Vesiculobullous lesions are short lived
- Once ruptured they leave painful erosions/ulcers
- Most lesions heal without scar formation
- Vesiculobullous lesions can be intraepithelial (intercellular) or subepithelial.

Diagnosis of vesiculobullous lesions

History

A detailed history is of paramount importance in the diagnosis of vesiculobullous lesions. Some aspects include:

- Onset of the blister formation
- Length of time the lesions have been present
- Whether they are cyclical
- Whether other parts of the body are involved
- Whether accompanied or preceded by constitutional symptoms
- Drug history.

Examination of vesiculobullous lesions

Examination of vesiculobullous lesions include:

- Clinical assessment with regard to their size/shape/number/color
- Presence or absence of special clinical signs such as Nikolsky's sign
- *Cytology*: Tzanck cells/viral inclusion bodies
- *Biopsy*: Histology/Immunofluorescence
- *Other*: Hematology/serology.

Classification of vesiculobullous lesions

Etiologic classification

Due to viral infections:

- Primary herpetic gingivostomatitis
- Herpes labialis (Recurrent HSV infection)
- Herpes zoster
- Herpangina
- Hand, foot and mouth disease.

Due to immunologic/genetic/uncertain etiology:

- Pemphigus
- Pemphigoid
- Dermatitis herpetiformis
- Linear IgA disease
- Erythema multiforme
- Epidermolysis bullosa
- Angina bullosa hemorrhagica.

Those of viral origin

Key features

- Mostly vesicular, multiple lesions
- Viral infections commonly involved include primary and secondary herpetic stomatitis, herpangina, herpes zoster and hand, foot and mouth disease
- These are preceded/accompanied by constitutional symptoms such as fever, malaise etc.
- Vesicles are mostly restricted to oral mucosa (except in Hand, Foot and Mouth disease and Herpes Zoster for example)
- Vesicles may be recurrent as in herpetic gingivostomatitis
- Clinically easy to diagnose, but serology is confirmatory.

Some details are given below:

Herpes simplex virus infections

Primary herpetic gingivostomatitis

Key features

- Herpes simplex virus Type I (occasionally Type II)

is the causative organism of primary herpetic gingivostomatitis

- This condition is common in childhood
- The disease is characterized by multiple vesicles on oral mucosa and gingivae which rupture leaving painful ulcers
- Constitutional symptoms such as fever, headache and submandibular lymph node enlargement are often present
- Primary herpetic gingivostomatitis is self limiting
- Ulcers form due to the rupture of vesicles which heal in about 10 days
- Circulating antibodies are present in this condition; these offer partial immunity
- The virus may become dormant in the trigeminal ganglion which, at a later period may get reactivated and cause recurrent secondary infection (e.g. herpes labialis)
- Treatment of the primary herpetic gingivostomatitis includes the use of antiviral agent (acyclovir) and anti-inflammatory agents for symptoms such as fever and pain.

Secondary herpes simplex infection

Key features

- This condition is caused by the reactivation of the virus dormant in trigeminal ganglion
- Multiple crops of tiny vesicles commonly appear on the lip or by the side of the ala of the nose (Fig. 13.1)
- Secondary herpes simplex infection is common in young adults
- Often patients may report tingling or burning



Fig. 13.1: Fluid-filled vesicles in secondary herpetic infection of the face. Note some blisters have ruptured leaving erosive areas

sensation on these sites prior to the appearance of vesicles (prodromal symptoms)

- Intraoral involvement is uncommon. When it occurs intra orally, attached gingivae and palate are the preferred sites
- Stress, exposure to strong sunlight, cold or immune deficiencies are often the factors responsible for the reactivation of the virus
- Vesicles in this condition rupture leaving painful ulcers
- Secondary involvement of the infection around fingernail sometimes occurs in dentists and nurses due to the direct contact with the active source of infection (if gloves are not used). This is called herpetic whitlow
- Treatment of secondary herpes simplex includes virus specific drugs such as acyclovir used topically or in severe cases systemically.

Varicella-zoster infections

Varicella (chickenpox)

Varicella or chickenpox is caused by Varicella-Zoster Virus (VZV). This is as a result of the primary infection. The secondary infection or reactivation of latent varicella-zoster virus is called herpes zoster or shingles.

Key features

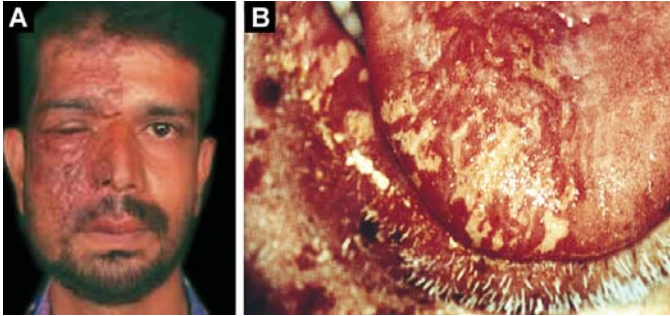
- Rash leading to painful vesicles on the trunk, face, and a few in the oral cavity are common findings of chickenpox. Skin lesions become pustular lesions and rupture leaving ulcers
- Chickenpox is common in childhood
- Constitutional findings such as fever, headache, chills and malaise are present
- Skin is extensively involved and pruritic
- Occasionally complications such as pneumonitis and encephalitis may occur
- If older individuals are affected the condition is usually much more severe
- Treatment of chickenpox includes the use of virus specific agents. Uses of corticosteroids are contraindicated in these patients
- Analgesics are useful in the relief of pain
- Recovery is achieved within several weeks.

Herpes zoster (shingles)

Herpes zoster is caused by the reactivation of the varicella-zoster virus which is dormant in the nerve ganglion

Key features

- Multiple vesicles occur unilaterally along the course of the sensory nerve (Fig. 13.2)



Figs 13.2A and B: Herpes zoster. Note the vesicles on right side of the face in a young person (**A**) and unilateral distribution of vesicles on the facial skin and the tongue in an elderly person (**B**). Most vesicles have ruptured leaving erosive areas

- These rupture and leave painful ulcers
- Common location of involvement includes the trunk, head, and neck. Rarely intraoral involvement is seen
- Affected individuals are usually adults
- Reactivation is uncommon in general population, commonly this occurs in immune suppressive states, malignancy or from irradiation treatment
- Prodromal symptoms such as pain prior to the occurrence of vesicles may be present
- Postherpetic neuralgia is a complication in about 10% of the patients with Herpes Zoster
- Treatment includes specific viral agents. Topical application of capsaicin often offers good results for postherpetic pain.

Coxsackie virus infections

Hand, foot and mouth disease (HF and MD)

The causative agent of hand foot and mouth disease is Coxsackie virus (Type A16). Airborne spread or

oro-fecal contamination from one individual to the other is the mode of transmission of infection.

Key features

- Infection occurs in epidemic or endemic proportions
- Children under the age of 5 years are commonly affected
- Low-grade fever, malaise, lymphadenopathy and sore mouth are the initial symptoms and signs.
- Oral lesions are vesicular which rupture leaving painful ulcers
- Ulcers of HF and MD exhibit a yellow fibrinous membrane surrounded by red halo
- Maculopapular lesions, which eventually become vesicular also, involve feet, toes, hands and finger. Ulcers and crusted lesions are eventually formed
- This is a self-limiting disease. Recovery is seen in about 2 weeks
- Treatment is usually symptomatic.

Herpangina

Herpangina is caused by the coxsackie (Types A1-6, A8, A10, A22) virus. Transmission of this disease is through contaminated saliva or feces.

Key features

- Commonly affects children
- Outbreaks of this disease usually occur in summer
- Symptoms and signs include malaise, fever, dysphagia and sore throat
- Vesicles appear on oral mucosa particularly on soft palate and tonsillar region
- These rupture and leave ulcers

- Recovery occurs within a week
- Symptomatic treatment is useful.

Vesiculobullous lesions of immunological background

This group of vesiculobullous conditions have a systemic background and hence may not be confined to oral mucosa alone. Oral lesions often may be the first clinical signs of the disease. Conditions in this category have serious health implications. A close consultation with patient's dermatologist or physician is required for management of these patients.

Key features of some of these conditions are listed below:

Pemphigus

Key general features

- An autoimmune mucocutaneous disorder
- Predilection for certain ethnic groups (Jews for example)
- Female preponderance in some studies
- Clinical types: *Pemphigus vulgaris*/*Pemphigus vegetans*/Familial form of Pemphigus (Haily Haily Disease)
- *Pemphigus vulgaris* is the commonest disorder with oral manifestations
- Oral lesions may precede skin lesions
- Fluid-filled bullae rupture leaving ragged painful erosions/ulcers (Fig. 13.3)
- Other mucosal surfaces may be involved
- Positive Nikolsky's sign
- Fluids, electrolytes and proteins are lost from the bullous disease, hence has serious threat to health if not treated early



Fig. 13.3: Pemphigus vulgaris. Note two tiny vesicles (arrows) on the gingival mucosa

- May be associated with other autoimmune diseases such as SLE, Thymoma and Myasthenia gravis.
- Biopsy and direct immunofluorescent studies are necessary
- Intraepithelial bullae and presence of Tzanck cells seen on histology
- Immunofluorescence is positive for intercellular antibodies
- *Antibodies seen are:* IgG and IgM
- Circulating autoantibodies are present in mucocutaneous form of the disease,
- Those with oral lesions alone may lack circulating autoantibodies

- In the absence of treatment disease can be fatal
- High doses of systemic corticosteroids are necessary: “High dose, short course”
- *Beware:* complications of long-term steroids
- Topical steroids are recommended for minor oral lesions
- Oral lesions need symptomatic treatment as well
- Patient’s physician is consulted.

Pemphigoid

An autoimmune disorder.

Two types: Mucous Membrane Pemphigoid and Bullous Pemphigoid.

Mucous membrane pemphigoid (MMP)

Key features

- Relatively common blistering disease
- Common among the elderly females
- Often oral lesions precede other mucosal lesions
- Involvement of the eyes may cause scarring leading to blindness
- Skin involvement is rare and minimal
- Occasionally mucosae of the nose, pharynx and genitalia are involved
- Desquamative gingivitis is common intraoral sign (Fig. 13.4)
- Nikolsky’s sign is positive
- Microscopically separation of the full thickness of the epithelium is seen at the Basement Membrane Zone (BMZ)
- Direct IMF shows linear binding of immunoglobulin IgG along BMZ
- Indirect IMF shows circulating autoantibodies IgG against BMZ antigens

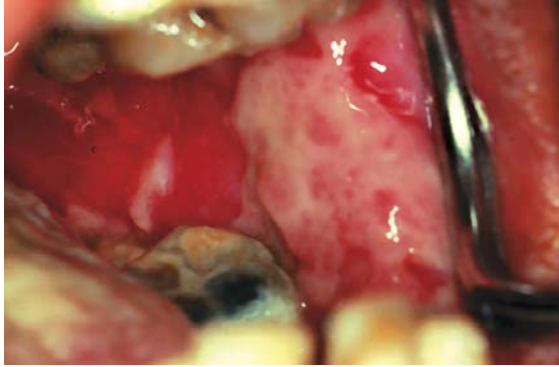


Fig. 13.4: Mucous membrane pemphigoid. Note the erosive red lesions on the buccal mucosa

Bullous pemphigoid (BP)

Key features

- BP predominantly involves skin
- Oral lesions are rare
- Disease is generally not fatal in the absence of treatment
- Can cause blindness when eyes are involved
- Autoantibodies against BMZ
- Systemic steroids are necessary
- Topical steroids (Betamethasone rinse for example) are useful for oral lesions.

Dermatitis herpetiformis (DH)

Key features

- Immunologically mediated rare mucocutaneous disorder
- Itchy, vesicular, herpetiform lesions on scapulae, arms, buttocks and legs
- Occasional oral vesicles/bullae

- Gluten sensitive enteropathy (coeliac disease) is present in these patients
- Deposition of IgA in granular pattern along the BMZ
- Treatment with Dapsone or Sulphonamides yield good results
- Gluten free diet is necessary.

Linear IgA disease

Key features

- Chronic autoimmune vesiculobullous disease of the skin
- Oral bullous lesions are occasionally seen
- Ocular lesions are common
- Direct IMF shows Linear IgA along the BMZ
- Systemic/topical steroids are used.

Vesiculobullous lesions of miscellaneous causes

Erythema multiforme (EM) and Stevens-Johnson syndrome

Key features

- Characterized by mucocutaneous inflammatory reactions arising as a consequence of immune complex mechanisms
- *Peak age incidence:* 20-30 years
- Male preponderance
- Majority are sequel to drug administration (sulfa for example)
- Some represent post-herpetic immune complex phenomenon
- May be associated with HIV infection/Lymphoma/Hepatitis Vaccination and SLE

- Skin lesions called Target or Iris lesions on palms and soles are characteristic
- Lips are always involved showing haemorrhagic brown-black crust (Fig. 13.5)



Fig. 13.5: Erythema multiforme. The lesions on the lip in this patient were preceded by blisters

- Intraoral sites may include buccal mucosae
- Severe form involving skin, mouth, esophagus and genitalia is called Stevens-Johnson syndrome
- The severe variant of EM without oral involvement is called Toxic Epidermal Necrolysis (TEN)
- Generally EM resolves within two weeks without treatment
- Systemic steroids may be useful
- Suspected drugs if known should be withdrawn
- Treatment is symptomatic
- If viral (HSV) association is suspected, acyclovir therapy is recommended.

Epidermolysis bullosa (EB)

Key features

- Genetic (Hereditary) and Acquired types of EB exist
- Bullae erupt at the site of minor trauma
- Children/young adults are commonly affected
- Skin over toes, fingers feet and elbows is commonly involved
- Oral blisters are seen in some cases
- Bullae rupture and ulcers heal with scar formation
- Hypoplastic teeth are occasionally seen
- Treatment includes systemic corticosteroids, Phenytoin, Vitamin E, Retinoids and Dapsone
- Avoidance of trauma is necessary.

Angina bullosa hemorrhagica (ABH)

Key features

- Large (1-3 cm) blood filled blister on the soft palate
- Unknown etiology
- Heals within a week
- Sometimes seen in asthmatics using inhalers.

Suggested reading

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CHAPTER

14

**Differential
Diagnosis of the
Developmental
Disorders of the
Teeth**

SR Prabhu

Learning objectives

After studying this chapter the student should be able to:

- Understand the basis of the occurrence of developmental disorders of the teeth
- List and clinically differentiate different types of the developmental disorders of the teeth.

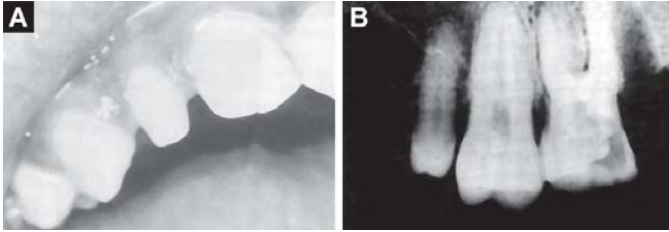
Introduction

In this chapter as space permits, several of the more common abnormalities and disturbances in tooth morphology and formation will be discussed. These abnormalities may be the result of genetic factors, environmental factors, or a combination of the two.

Anomalies related to the size of teeth**Microdontia and macrodontia**

Variations in the size of teeth may be localized, involving one or a few teeth, or generalized, involving the entire dentition. The variations may be true or may be relative, i.e., normal or near normal size teeth in either larger or smaller jaws. A localized variation in size is more common in both micro- and macrodontia.

- Microdontia is much more common than macrodontia with microdontia involving only a single tooth being a rather common condition
- The maxillary lateral incisor and the third molar are the most frequently observed microdonts (Figs 14.1A and B)
- The incisally tapering maxillary lateral incisor with its conical crown form has been termed the 'peg lateral'. This disorder is often familial.



Figs 14.1A and B: **A.** Microdontia of maxillary lateral incisor and **B.** Maxillary third molar

Anomalies related to the shape of teeth

Gemination

Gemination results either from the splitting of a tooth germ during development or from the fusion of a normal tooth bud with one from a developing supernumerary tooth.

- The permanent maxillary incisors and the primary mandibular incisors are most often affected by this anomaly which shows no particular sex predilection
- The affected tooth usually shows two completely or incompletely separated crowns which have one shared root and root canal. On occasion the anomaly may be bilateral.

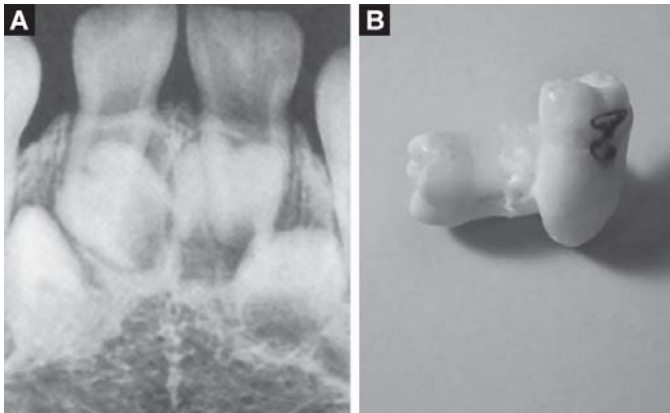
Gemination of teeth may be distinguished from fusion by the fact that in gemination a full complement of teeth is present while in fusion a tooth is usually missing.

Fusion

Fused teeth arise through the union of two or more juxtaposed normal tooth germs. The junction is at

the level of the dentine and, depending upon the stage of tooth development at the time of fusion, it may be complete or incomplete (Fig. 14.2A).

- If contact occurs before calcification begins, a single large tooth may result from the complete union of the tooth germs involved. Hereditary pattern is often encountered
- If contact occurs after a portion of the crown has been completely formed, there may be a union of the roots only.



Figs 14.2A and B: **A.** Fusion between deciduous mandibular left central and lateral incisors and the right central and lateral incisors, **B.** Concrecence

This anomaly is reported to be more common in the primary dentition with the incisors being the most frequently affected in both dentitions.

Concrecence

In concrecence there is union of two or more juxtaposed teeth by cementum (Fig. 14.2B).

- Concrescence may occur before or after the teeth erupt
- The permanent molars are reported to be affected by this anomaly more frequently than the other teeth
- The diagnosis of concrescence can usually be established by radiological examination.

Dilaceration

Dilaceration refers to an angulation, sharp bend, or curve in the root or crown of a developed tooth (Fig. 14.3).

- Dilaceration results from trauma or some defect in tooth development which alters the angulation of the tooth germ during root and crown formation
- Both dentitions manifest this anomaly and it is by no means uncommon
- This anomaly is quite evident upon radiological examination.

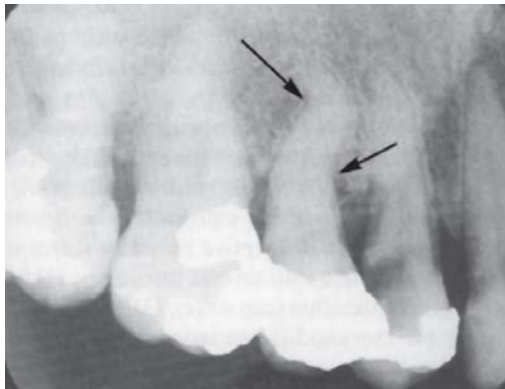


Fig. 14.3: Dilaceration seen in the root of second pre-molar. Arrows show angulation in two places

Talon cusp

Talon cusp is a structure resembling an eagle's talon. This is seen as a cusp projecting lingually from the cingulum area of a maxillary or mandibular permanent incisor.

Dens invaginatus (dens in dente)

Dens invaginatus is a developmental anomaly which represents a defect in morphological development arising as a result of an invagination in the surface of a tooth crown before calcification has occurred.

- Increased localized external pressure, focal growth retardation, and focal growth stimulation have each been proposed to cause this oral anomaly
- The permanent maxillary lateral incisors are the teeth most frequently involved and bilateral involvement is common
- A radicular variety often involving molar teeth has also been reported
- A coronal invagination may be limited to the crown or may extend to the root tip
- Clinically, the affected tooth crown may exhibit a somewhat bulbous appearance or be of normal shape. In the latter the presence of an enlarged lingual pit may be the only external manifestation of a dens in dente.

In radiographs the lesion is recognized as a 'pear-shaped' invagination of enamel and dentine with a narrow constriction at the tooth surface. Deep invaginations closely approximate the pulp.

Dens evaginatus (evaginated odontome)

Dens evaginatus is a developmental anomaly that appears clinically as an accessory cusp or enamel-

covered tubercle projecting from the occlusal surface of a tooth. Most cases involve premolar teeth. Dens evaginatus may be unilateral or bilateral.

Clinically, dens evaginatus is easily recognized. This extra cusp may cause problems similar to those encountered with talon cusp.

Taurodontism

The term taurodontism is used to describe the peculiar dental anomaly of 'bull teeth' in which the body of the tooth is enlarged at the expense of the roots. This dental defect involves a marked elongation and enlargement of the pulp chamber (Fig. 14.4).

- Clinically, the crowns of affected teeth appear normal; the defect only being observable on dental radiographs
- Either dentition may be involved, but the condition is more common in the permanent teeth
- The molar teeth are more frequently involved and one or more teeth, unilaterally or bilaterally, may exhibit this anomaly.



Fig. 14.4: Taurodontism seen in teeth

Accessory roots

Accessory roots may be encountered on any tooth type including premolars and canines.

Clinical and/or radiographic examination will easily enable the clinician to recognize these entities.

Shovel-shaped incisors

The term shovel-shaped describes a morphological anomaly of the crowns of incisor teeth. The shovel shape is manifested by the prominence of the mesial and distal marginal ridges which enclose a central fossa on the lingual surface of incisor teeth. Shovelling is more common in the maxillary teeth than in the mandibular incisors.

Anomalies involving the number of teeth

Anodontia

Congenital absence of teeth results in true anodontia. This may be either total (oligodontia) or partial (hypodontia).

- Total anodontia, in which all the teeth are missing, is a rare condition and when observed is frequently associated with hereditary ectodermal dysplasia
- Total anodontia may involve both the primary and permanent dentitions
- Hypodontia, involving one or more teeth also called partial anodontia is a relatively common condition (Fig. 14.5)
- Hypodontia is occasionally encountered in the deciduous dentition. In this dentition it is the maxillary lateral incisor which is most frequently missing
- Hypodontia due to destruction of tooth germ tissue may occur in children who receive high doses of

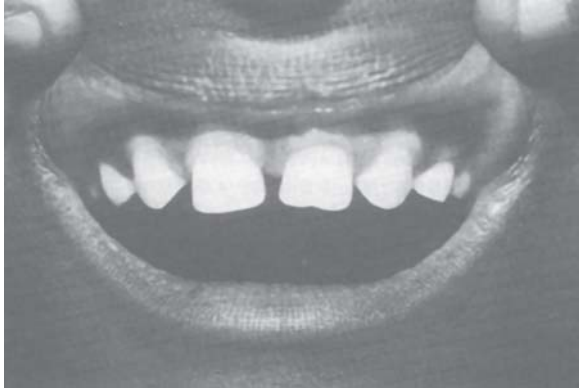


Fig. 14.5: Partial anodontia. Note bilateral congenitally missing maxillary lateral incisors

therapeutic radiation to the jaws at an early age for conditions such as malignant neoplasms

- In addition to hereditary ectodermal dysplasia, anodontia (total or partial) is associated with a variety of other hereditary disorders including chondroectodermal dysplasia, and Rieger's syndrome.

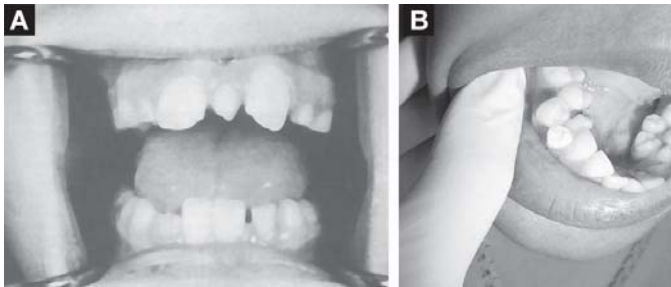
In evaluating missing teeth in patients it should be determined if the teeth have been extracted, creating an 'induced' or 'false' anodontia. The occlusion may be defective and/or the esthetics may not be pleasing when teeth are congenitally missing.

Supernumerary teeth

Supernumerary teeth (hyperdontia) are relatively common in the permanent dentition.

- The most common supernumerary tooth is the mesiodens

- A mesiodens is a small tooth with a conical crown and a short root (Fig. 14.6A)
- Mesiodens are most often found as solitary entities. However, occasionally, paired mesiodens are encountered
- Mesiodens occur in the midline of the anterior maxilla. They may erupt or remain unerupted. An unerupted mesiodens may be responsible for a diastema between the maxillary central incisors or for the non-eruption or delayed eruption of a maxillary central incisor
- Supernumerary premolars are also common (Fig. 14.6B)
- Supplementary fourth molars (paramolars), maxillary lateral incisors, mandibular incisors, and premolars are sometimes seen in patients' mouths
- Supernumerary fourth molars are most often seen in the maxilla and appear as small, atypical teeth. On the other hand supplementary maxillary lateral incisors, mandibular incisors, and premolars



Figs 14.6A and B: **A.** Mesiodens seen in between the two maxillary central incisors and **B.** Supernumerary lower premolars

almost invariably appear as normal teeth. Supernumerary primary teeth are rare.

Predeciduous and postpermanent dentitions

Occasionally infants are born with structures in their mouths, usually in the anterior region of the mandible, which appear to be erupted teeth. On occasion, true primary teeth, the natal teeth have erupted by the time of birth. However, in many cases the structures seen probably represent dental lamina cysts of the new-born rather than natal teeth or predeciduous teeth. It is important to recognize a natal tooth as a true primary tooth since obviously these teeth should not be removed.

On occasion a postpermanent or third dentition has been reported. In the majority of these cases, the newly erupted teeth are only the result of the delayed eruption of retained, embedded, normal, or supernumerary teeth.

Anomalies related to alterations in the structure of teeth

Amelogenesis imperfecta

Hereditary defects of enamel, usually unassociated with any other generalized defects, are known as amelogenesis imperfecta. These developmental defects are entirely ectodermal in nature and derive from abnormalities in amelogenesis.

Amelogenesis imperfecta is classified into three basic types corresponding to the three stages of normal enamel development:

- A hypoplastic type when there is defective formation of the enamel matrix during the formative stage
- A hypocalcification (hypomineralization) type when defective mineralization of the matrix occurs during the calcification stage
- A hypomaturation type when the enamel crystallites do not enlarge and mature during the maturation stage of enamel formation.

The hypoplastic type is noted when the enamel has not formed to a full thickness on newly erupted teeth.

- If the enamel is so soft that it is removed during dental prophylaxis procedures, the amelogenesis imperfecta is classified as the hypocalcified type.
- The hypomaturation type is composed of enamel which can be pierced by an explorer under firm pressure and can be chipped away from the underlying dentine.
- In amelogenesis imperfecta all teeth from both dentitions are affected to some extent.
- Whereas, in some cases, the teeth may appear clinically normal, in others the teeth exhibit marked signs of enamel abnormality.
- The teeth may or may not show discoloration and, if present, the color may range from yellow to dark brown to black.
- Affected teeth may have no enamel and, if enamel is present, its texture ranges from hard to soft.
- In some types of amelogenesis imperfecta the enamel is smooth, while in others it is wrinkled or grooved.
- The enamel may be chipped away and/or severely abraded.

- Radiographically, the enamel may be absent or thin and in certain cases have approximately the same radiodensity as the dentine.

Amelogenesis imperfecta must be differentiated from environmental enamel hypoplasia and/or hypocalcification and dentinogenesis imperfecta.

Enamel hypoplasia

Severe damage to ameloblasts during amelogenesis will result in defective or incomplete formation of the organic matrix of enamel which is denoted by the term enamel hypoplasia. Two basic types of enamel hypoplasia exist:

- A hereditary type discussed as amelogenesis imperfecta
- An environment type.
 - Clinically, enamel hypoplasia may manifest as focal or generalized areas of enamel deficit or roughness on tooth surfaces, as discrete pits or arrays of pits on the crown, or as grooves on the coronal surface
 - A number of conditions may be capable of producing environmental enamel hypoplasia. Nutritional deficiencies, exanthematous diseases, hypocalcemia, birth injuries, local infection or trauma, premature, and Rh hemolytic disease are all capable of interfering with the various phases of amelogenesis and producing enamel hypoplasia
 - However, in many patients no cause for enamel hypoplasia is found. Enamel hypoplasia is associated with congenital syphilis and excessive fluoride intake

- Local infection or trauma is probably the most frequent cause of enamel hypoplasia. Focal periapical infection from a carious primary tooth or trauma to a primary tooth may cause damage to the ameloblasts of a developing succedaneous permanent tooth
- The crown of the affected tooth (the so-called Turner's tooth) is yellow to brown in color and pitted (hypoplastic) to chalky (hypocalcified) at the site of involvement
- On occasion, many teeth in a patient may show pitting or roughness as well as color changes. Febrile childhood illnesses, hypocalcemia, or birth injuries may cause this type of distribution of enamel hypoplasia.

Enamel hypoplasia of congenital syphilis

Congenital syphilis is contracted in utero from a mother who has an active infection of syphilis. Virtually any organ or tissue may be involved. Frontal bossing, a short maxilla, a high-arched palate, saddle nose, enamel hypoplasia, rhagades, and saber shin are all included among the structural anomalies seen in congenital syphilis. The classic dental changes involve the incisor, generally the maxillary, and the permanent first molars. Hutchinson's triad, which includes enamel hypoplasia of the teeth, interstitial keratitis, and eighth cranial nerve deafness, is considered pathognomonic for congenital syphilis.

Enamel hypoplasia due to fluoride intake

Mottling of the enamel may occur when fluoride levels in the drinking-water exceed one part per million. In regions of the world where the natural drinking water

supplies have a high fluoride content, dental fluorosis is common (Fig. 14.7).

- Teeth affected by fluorosis vary considerably with regard to the severity of enamel changes seen. The

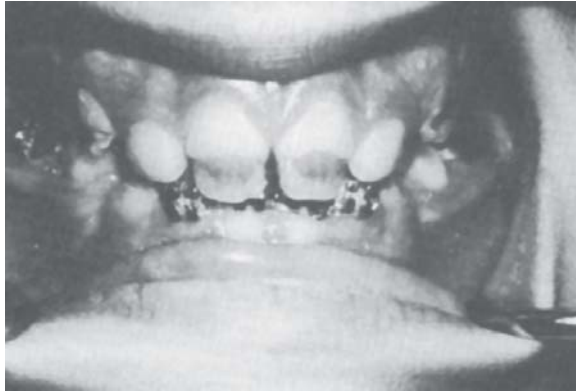


Fig. 14.7: Enamel hypoplasia due to excessive fluoride intake during the formation of crown central incisors

latter include hypocalcification and hypoplasia

- The crown of affected teeth may appear brown to black with extensive flaking or may appear chalky and opaque. All teeth exposed to excess fluoride during their development are involved.

Enamel hypoplasia due to nutritional deficiencies and exanthematous fevers

Since the ameloblasts are among the most sensitive groups of cells in the body, any serious nutritional deficiency or exanthematous disease is potentially capable of producing injury to these cells and thus causing enamel hypoplasia.

- In mild hypoplasia related to these two causes, there may be only a few small pits, grooves, or fissures of the surface enamel
- A more serious insult may result in enamel exhibiting rows of deep pits arranged horizontally across the tooth surface
- There may be only one row of such pits or several rows indicating a series of injuries. With a serious, prolonged disturbance in the function of the ameloblasts, large portions of the enamel may be missing
- Enamel hypoplasia can occur only during the formative stage of enamel development and thus, by knowing the chronological development of the teeth, one can determine from the location of the defect on the particular teeth the approximate time at which the insult occurred
- Numerous studies have shown that enamel hypoplasia commonly occurs in patients with rickets during the time of tooth development. Deficiencies of vitamins A and C have also been implicated.

Exanthematous diseases such as measles and chickenpox can also occasionally cause enamel hypoplasia.

Dentinogenesis imperfecta (Hereditary opalescent dentine)

Hereditary opalescent dentine is a developmental anomaly of dentine which is inherited as an autosomal dominant trait.

There are three recognized types of dentinogenesis imperfecta:

- *Type I:* This type of dentinogenesis imperfecta always occurs in families with osteogenesis imperfecta, an association well recognized for many years
- *Type II:* This type of dentinogenesis imperfecta is not associated with osteogenesis imperfecta
- *Type III:* The 'Brandywine' type is uncommon. Multiple pulp exposures in the primary teeth give a clinical characteristic of this type not seen in the other two types.

Both dentitions are affected by the disorder resulting in teeth ranging in color from grey to yellowish brown. The teeth exhibit an unusual characteristic translucent or opalescent hue. The overlying enamel has a tendency to chip and erode especially on incisal and occlusal surfaces (Fig. 14.8).

In the posterior teeth the crown form has a tendency to be bulbous and the roots short and conical. Partial



Fig. 14.8: Dentinogenesis imperfecta. Opalescent hue of the teeth and chipping away of occlusal and incisal surfaces is seen

to complete precocious obliteration of the pulp chambers and root canals by the continued formation of dentine is a striking radiological feature of dentinogenesis imperfecta.

Dentine (dental) dysplasia

Dentine dysplasia has been separated into two types: Type I is a radicular dentine dysplasia while type II is a coronal type.

- Type I is reported to be more common. In type I radicular dentine is affected with the coronal portion of the tooth being morphologically normal. In this condition the tooth roots are rudimentary (rootless teeth). Occasionally this leads to spontaneous exfoliation. The root canals are obliterated and the malformed pulp chambers appear as horizontal crescents. Periapical pathology, often of unknown origin, is commonly found at the apex of the teeth
- In dentine dysplasia type II, the primary teeth are translucent and amber-grey in color. This feature, when coupled with the radiographic evidence of pulp and root canal obliteration seen in this condition, gives the teeth an appearance similar to those of dentinogenesis imperfecta. The permanent teeth exhibit abnormally large coronal pulp chambers often described as 'thistle-tube' in appearance.

Regional odontodysplasia (ghost teeth)

Regional odontodysplasia is a localized oral developmental anomaly restricted to a single tooth or a group of contiguous teeth. The teeth involved either exhibit delayed eruption or do not erupt at

all. On radiographic examination, both enamel and dentine formation appear deficient, highlighting dilated pulp chambers. The term 'ghost' teeth comes from the faint, radiographic appearance of the affected teeth.

Suggested reading

1. Regezi JA, Sciuba J. Abnormalities of teeth: In Oral Pathology: Clinical and Pathologic Considerations, WB Saunders. Company, Philadelphia 1993;494-517.
2. Sawyer DR. Developmental disorders of the teeth. In Prabhu SR, et al (Eds): Oral Diseases in the Tropics, Oxford University Press 1992;543-48.

CHAPTER

15

**Differential
Diagnosis of the
Acquired
Disorders of
the Teeth**

SR Prabhu

Introduction

Acquired disorders involving teeth are extremely common. These are essentially posteruptive disorders and occur due to oral environmental factors. Majority of the disorders however do not pose major diagnostic problems to the clinician.

Important disorders in this group of acquired disorders of teeth include:

- Dental caries
- Tooth attrition
- Tooth abrasion
- Tooth erosion
- Tooth fracture
- Tooth discoloration

Discussion in the following paragraphs is kept brief and up to the point with a view to assisting the student in the diagnosis. Further details can be obtained from standard textbooks on Clinical Dentistry.

Dental caries

Dental caries is a very common disease affecting the teeth of primary and permanent dentitions. The disease is essentially “a localized, post-eruptive pathologic process of external origin involving softening of the hard tooth tissue and proceeding to the formation of a cavity” (WHO).

Dental caries has been classified in a number of ways.

- According to the location of caries on the individual tooth:
 - Pit and fissure caries
 - Smooth surface caries.
- According to the rapidity of the carious process:
 - Acute dental caries

- Chronic dental caries
- Arrested dental caries.
- According to whether the lesion is new or recurrent:
 - Primary dental caries
 - Recurrent (secondary) dental caries.

Volumes have been written on the etiology of dental caries. Essentially it is due to the demineralization of tooth structure by the acids that are produced in the oral environment due to the action of oral acidogenic/aciduric bacteria on the carbohydrates found in the food and drinks. Dental plaque is a major contributor to this process.

Clinically, the diagnosis of fully developed carious lesions is not difficult. However, insipient lesions of dental caries often present diagnostic difficulties.

Examination of carious lesions

Clinically both visual and exploratory methods are used to recognize carious lesions.

Color changes and translucency of tooth surfaces are observed in the carious process. These include:

- Chalky white areas of demineralization. Chalky white areas of incipient caries are often mistaken for hypoplastic lesions
- Grayish-white discoloration of marginal ridges
- Grayish-white color extending from margins of restorations
- Open carious lesions with yellowish-brown to dark brown color (Fig. 15.1)
- Dull, flat white opaque areas seen under direct light showing loss of translucency
- Dark shadow on a proximal surface as shown by transillumination



Fig. 15.1: Occlusal caries as a brown lesion

- Typical cervical carious lesion is a crescent-shaped cavity that often shows a roughened chalky area (Fig. 15.2)
- Recurrent caries is seen adjacent to existing restorations
- Arrested caries is characterized by a large open cavity in which there is a lack of food retention and the carious lesion takes on a brown stained polished hard surface (Fig.15.3)
- Bite wing radiographs reveal demineralized carious lesions on the proximal surfaces which are otherwise inaccessible clinically
- *Rampant caries:* When several teeth are involved by active caries it is called rampant caries (Fig. 15.4)



Fig. 15.2: Cervical caries
Courtesy: Dr R Rafeek



Fig. 15.3: Arrested caries

- On radiographs the phenomenon of ‘cervical burn out’ at the cervical regions of the teeth should not be mistaken for carious lesions.



Fig. 15.4: Rampant caries involving all teeth in a very poor state of oral hygiene

A detailed discussion on different aspects of dental caries is beyond the scope of this chapter.

Tooth attrition

Attrition is the physiologic wearing away of a tooth as a result of tooth-to-tooth contact as in mastication or in bruxism.

- Attrition is usually found on occlusal, incisal and proximal surfaces. Attrition is also regarded as an aging process as this condition is common among the elderly
- Initial sign of attrition is the appearance of a small polished facet on a cusp tip or flattening of an incisal edge
- In advanced attrition enamel has completely been worn away. This often results in a yellow or brown staining of the exposed dentin. Stain is usually derived from food or tobacco. In some instances teeth are worn down nearly to the gingivae

- Radiographically pulp-chamber and canals may be narrowed due to the deposition of secondary dentin
- Advanced cases of attrition should be differentiated from dentinogenesis imperfecta. The latter however is a genetically derived condition and is seen in the younger age group.

Tooth abrasion

Abrasion is a pathologic wearing away of tooth substance through some abnormal mechanical process such as faulty brushing.

- Usual sites of abrasion are the exposed root surfaces of the teeth
- On occasion abrasion can be seen on other surfaces such as incisal or proximal surfaces
- Trauma caused by injudicious use of tooth brush often results in a V-shaped ditch at the cemento-enamel junction with some gingival recession
- Other examples of abrasion include defects derived from habits or occupation of the patient. Habitual opening of the bobby pins with teeth may result in producing a notch of the incisal edge of the maxillary central incisor
- Carpenters, shoe makers and tailors who hold nails, pins or needles in between their maxillary and mandibular incisors develop abrasion of the tooth surfaces as notches
- Improper use of tooth pick can produce abrasion of the proximal root surfaces of the teeth.

Tooth erosion

Loss of tooth substance by a chemical process from a non bacterial source is called erosion.

- Lesions of erosion occur commonly on the labial and buccal surfaces of teeth. They are smooth, shallow, highly polished scooped out depressions on the enamel surfaces-close to cemento-enamel junction
- Those who drink large quantities of acidic carbonated beverages or lemon juice or suck on citrous fruits may exhibit lesions of erosion on the labial surfaces of the teeth
- In patients with chronic vomiting lingual surfaces of anteriors may exhibit erosion
- In anorexia nervosa in which induced chronic vomiting is common, lesions of erosion are often seen on the lingual surfaces of teeth
- Erosion of the teeth is also common among persons working in industries involving the use of acids. Battery manufacturers, galvanizing industry workers and soft drinks workers are among those who are prone to exhibit dental erosion.

Lesions of erosion should be differentiated from those of abrasion.

Tooth fractures

Trauma to the face often results in tooth fracture.

Fractured teeth exhibit the line of fracture in three forms: Horizontal, diagonal and vertical (Fig. 15.5).

Radiographic evidence of the fracture is usually present. Fractures of teeth are classified as (WHO):

- Fracture of enamel only
- Fracture of crown of tooth without pulpal involvement
- Fracture of crown of tooth with pulpal involvement.



Fig. 15.5: Fracture of the upper left central incisor involving enamel

- Fracture of root of tooth
- Fracture of crown and root of tooth with or without pulpal involvement
- Fracture of tooth unspecified
- Luxation (dislocation) of tooth
- Intrusion or extrusion of tooth
- Avulsion of tooth.

A proper history would reveal the cause of fracture. Crack in the tooth is often difficult to diagnose on X-rays alone.

Dental stains and discolorations

Stains and discolorations of teeth are exogenously and/or endogenously derived.

Exogenous stains can be removed by tooth brushing and/or polishing tooth surfaces.

Endogenous stains cannot be removed by scaling and polishing.

- Most frequently observed exogenous stains include those derived from tobacco, food, drinks or from chromogenic bacteria
- The color of stains can vary yellow, green, black, and brown are commonly occurring stains
- Betel leaf stains are dark mahogany brown in color.

Endogenous stains are seen in different clinical conditions.

- These include, erythroblastosis fetalis, tetracycline insult during tooth formation, amelogenesis imperfecta, dentinogenesis imperfecta and advanced dental fluorosis. In the latter three instances defects in tooth surface are also evident.
- Proper history and clinical examination assist in the diagnosis of these conditions.

A detailed discussion on these conditions is beyond the scope of this chapter.

Suggested reading

1. Birnbaum W, Dunne SM. Oral Diagnosis: The Clinician's Guide. Wright: Oxford 2000.
2. Rigezi JA, Sciubba J. Oral Pathology: Clinical-Pathologic Correlations (2nd edn). WB Saunders Co, Philadelphia 1993.

CHAPTER

16

**Differential
Diagnosis of
Developmental
Disorders of
Oral Mucosa**

SR Prabhu

Developmental lesions of the oral mucosa are common. Some of these lesions are symptom free. When patients become aware of their presence they become unduly concerned. Often dentist is the first person to identify these lesions. Although the majority of developmental lesions are harmless, a few entities may possess malignant potential. Correct diagnosis of these is therefore essential and differential diagnosis of these lesions is not always easy.

The developmental lesions of the oral mucosa can be classified in many ways. A classification based on the tissue of origin is proposed here. Clinical classification can be offered on the basis of presentation of oral lesions such as those with white, yellow, red, nodular and other features.

Classification

Epithelial lesions

- White sponge nevus
- Darier-White's Disease
- Dyskeratosis congenita
- Hereditary benign intraepithelial dyskeratosis
- Pachyonychia congenita
- Pigmented nevi-Intradermal (Intramucosal)
 - Junctional
 - Compound
 - Spindle cell/Epithelioid cell
 - Blue nevus.
- Peutz Jegher's syndrome.

Connective tissue lesions

- A. Lesions with proliferations of cells and structures normally present (Hamartomas):
 - *Blood vessels*
 - Haemangioma
 - Encephalo trigeminal angiomatosis (Sturge-Weber Syndrome)
 - Hereditary haemorrhagic telangiectasia
 - *Lymphatic vessels:*
 - Lymphangioma
 - Cystic hygroma
 - *Nerves:*
 - Multiple neurofibromas (neurofibromatosis)
 - *Fibrous connective tissue:*
 - Fibromatosis gingivae
 - *Lymphoid tissue:*
 - Lingual tonsil
- B. Lesions with normal tissue in ectopic position:
 - Fordyce granules
 - Lingual thyroid
- C. Persistence of embryonic structures and/or failure of their proper development:
 - Congenital lip and commissural pits and fistulas
 - Double lip
 - Double frenulae
 - Ankyloglossia
 - Clefts-lip:* with or without palatal involvement
 - Tongue
 - Uvula
 - Median rhomboid glossitis
 - Benign migratory glossitis
 - Thyroglossal duct cyst

- Congenital epulis
 - Dermoid cyst
- D. Miscellaneous mucosal lesions with possible developmental pathology:
- Fissured tongue
 - Cheilitis granulomatosa
 - Cheilitis glandularis
 - Focal epithelial hyperplasia

Briefly the above clinical entities are discussed in the following paragraphs:

Epithelial lesions

White sponge nevus

This condition is also known as Cannon's disease, Congenital leukokeratosis mucosa oris, Hereditary leukokeratosis and White folded gingivostomatitis.

- Is an autosomally dominant inherited benign keratotic lesion of the mucosal surfaces
- Is uncommon but not rare
- Affects both sexes equally
- Is seen either congenitally or at puberty
- Is generally symptomless
- *Involves oral mucosa:* any site. Common site is buccal mucosa followed by tongue, floor of the mouth, palate and labial mucosa. May involve entire oral mucosa
- Other mucosal sites include nasal, pharyngeal, oesophageal, rectal, anal and genital mucosa
- Is a white lesion with variable severity. May appear either as diffusely keratotic lesion with spongy or wrinkled surface; or white lesion with opalescent appearance.

- *Must be differentiated from*
 - Leukoedema
 - Leukoplakia, and
 - Lichen planus and other related white lesions. History and clinical appearance should make the distinction clear (along with microscopic appearance).
- Remains symptomless throughout life
- No treatment is usually necessary.

Darier-White's disease

- Is also known as Darier's disease and Keratosis follicularis (a misnomer?)
- Is a dominant autosomally transmitted disease characterized by hyperkeratotic papules occurring on mucosal and cutaneous surfaces
- Is very rare
- Affects both sexes equally
- Is usually first seen in childhood
- Is usually asymptomatic
- Cutaneous lesions are seen on—Forehead, scalp, neck, chest, back and axillae and groin
- Mucous membrane—Oral (50%), rectal, anal and genital mucosal surfaces. Oral cavity—gingiva and tongue
- Small firm red papules initially; grayish brown later
- Ulceration of the same may occur. Crust is present on cutaneous lesions. Oral lesions are smaller with verrucous white or cobblestone appearance
- Differential diagnosis includes other inherited white lesions of oral mucosa especially those with cutaneous involvement such as Pachyonychia congenita and dyskeratosis congenita

- Malignant transformation is not recorded
- Large doses of Vitamin A are recommended, but this treatment method has its own morbidity.

Dyskeratosis congenita

- Is a recessively transmitted (probably), disease characterized by changes in nails, skin and mucous membranes
- Is rare
- Shows predilection for males
- Is seen in individuals 5-50 years of age
- Affects nails, skin and oral, anal, renal and GI tract mucosa
- Nails—become dystrophic and shed off
This is initial sign—starting after the age of 5 years.
- Skin—brown pigmentation on trunk, thighs and neck. This follows changes in the nails.
- *Oral lesions:*
 - Vesicles and ulcerations followed by white patches of necrotic epithelium between 5-14 years of age
 - Ulcerations and red patches between 14-20 years of age
 - Erosive white patches and occasionally carcinoma between ages of 20-30 years.
- Though not specific, haematologic examination for anaemia, leucopenia, thrombocytopenia, etc. (that of Fanconi Syndrome) are carried out
- White lesions possess malignant potential. High incidence of oral cancer in young affected individuals has been reported
- No treatment is known. Periodic examination is essential.

Hereditary benign intraepithelial dyskeratosis

- Is an autosomal dominant disease characterized by white, symptomatic thickened plaques on the oral mucosa and hyperemic perilumbar bulbar conjunctivae
- Originally seen in American Indian and Negro ancestry. Not necessarily restricted to this group. Is a rare condition
- Has no sex predilection
- Usually appears in infancy
- Presents photophobia
- Oral lesions include—white spongy lesions of buccal mucosa. Folded appearance and opalescent hue may be present
- Eye lesions include—conjunctivae or congested and white plaques overlying the cornea
- Cytology in addition to histology is helpful. Papanicolaou stain reveals dyskeratotic cells
- Differential Diagnosis includes White sponge nevus and other non-inheritable white lesions of oral mucosa (Leukoplakia—in particular)
- Persists throughout life. Malignant change has not been reported
- No treatment is indicated.

Pachyonychia congenita

- Is an autosomally dominant hereditary disease, characterized by the involvement of finger and toe nails, palms and soles, cornea and oral mucous membrane
- Is a rare condition
- Has no known sex predilection
- Is congenital or during neonatal period

- Is symptomless
- Involves nails, palms and soles and posterior buccal mucosa
- Manifests as thickening of subungual keratin of the nails, hyperkeratotic calluses of palms and soles, follicular keratosis of knees and elbows and white opaque patches of buccal mucosa. Natal teeth have been also reported
- Differential diagnosis includes white sponge nevus. Hereditary benign intraepithelial dyskeratosis and Leukoplakia
- No malignant transformation has been recorded.
- Persists throughout life
- No treatment is necessary.

Melanotic macule and pigmented nevi

- Melanotic macule is a flat pigmented lesion that sometimes occur on the lip as shown in Figure 16.1. This lesion is formed because of excessive amount of melanin pigment formed by normal melanocytes located in epithelium

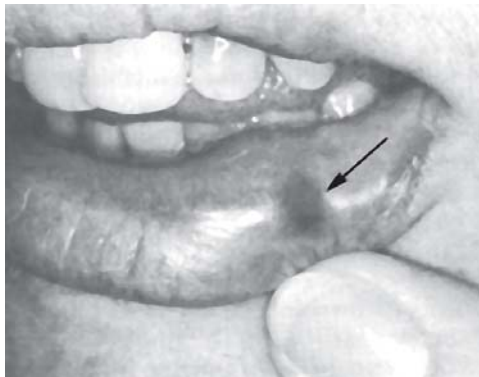


Fig. 16.1: Labial melanotic macule (arrow)

- Pigmented nevi are also known as pigmented mole and cellular nevus. These are formed by nevus cells located in submucosa
- Pigmented nevi are congenital developmental tumor like malformations of the skin or mucous membranes, usually containing melanin pigment. Common types are—
 - Intradermal nevus (common mole)
 - Junctional nevus
 - Compound nevus
 - Spindle cell/Epithelioid cell and
 - Blue nevus.
- They are common in skin but uncommon in oral cavity
- Show no sex predilection
- Occur at any age. Majority of blue nevi however are present at birth or appear in early childhood.
- They are asymptomatic
- All the four types occur in skin and mucous membrane. Intraoral sites any, but most frequently anterior gingivae, lips and palate (Fig. 16.2)



Fig. 16.2: Mucosal naevus on the palatal mucosa

- Generally all cellular nevi are well circumscribed, smooth, flat or raised pigmented (occasionally unpigmented) lesions. Intradermal type (intra-mucosal) is usually firm, nodular. Compound nevus is firm and raised, nodular (as intradermal type), and blue nevus is dark blue in color, which can be moved over the sub-mucosal structures. These lesions are discrete lesions and not patchy (as is the racial pigmentation seen in some patients)
- Differential includes other focal pigmented lesions, e.g. amalgam tattoo, ephelis (freckle) and melanoma
- Although these have a benign course, mucosal nevi are surgically removed as a prophylactic measure, because of the constant irritation of the area during brushing and eating
- If a pigmented nevus ulcerates, or shows rapid enlargement in size, or becomes painful, it must be viewed suspiciously. Melanomas, on rare occasions, may arise from pre-existing nevi.

Peutz-Jegher's syndrome

- This is also known as hereditary intestinal polyposis with melanin pigmentation
- Is an autosomally dominant disease characterized by macular hyperpigmentation of the lips (Fig. 16.3), buccal mucosa, and/or fingers with intestinal polyposis
- Condition is rare.
- Shows no sex predilection
- May be present at or just after birth. Distinct at puberty

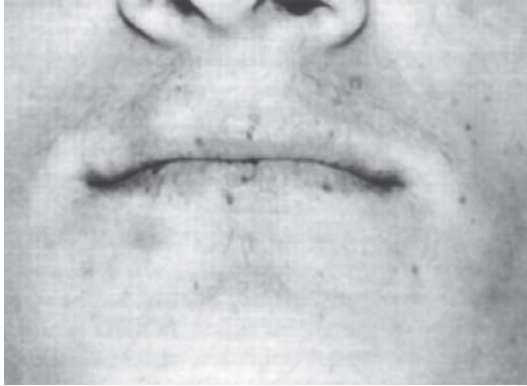


Fig. 16.3: Peutz-Jegher's syndrome showing perioral pigmentation

- Patient complains colicky, intermittent abdominal pain
- Pigmentation is seen about the facial orifices, buccal mucosa, labial mucosa and hands and feet. Polyps are found in small intestine
- Oral pigmentation. Small brown specks of about 1 to 5 mm in diameter occur most frequently on buccal mucosa, followed by gingivae and hard palate. On the face they are around nostrils, lips and eyes
- Differential diagnosis includes all conditions of oro mucosal pigmentation. GIT findings differentiate.
- Oral/Paraoral pigmentation is benign and persists throughout life. Intestinal polyposis may have a low malignant potential, hence an early diagnosis is important
- Surgical removal of polyps as a prophylactic measure is debatable.

Connective tissue lesions

A. Lesions with proliferations of cells and structures normally present

Hemangioma

- Is a vascular nevus
- Is characterized by the proliferation of blood vessels (Fig. 16.4)



Fig. 16.4: Haemangioma

- Is common in head and neck region (56%)
- Shows female to male ratio as 65:35
- Is present at birth or appear in early childhood.
- Is asymptomatic unless ulcerated
- Intraoral sites include—tongue, lips, buccal mucosa and palate
- Flat or raised lesions of mucosa. Circumscribed red or blue lesion, which blanches on pressure
- Differential diagnosis includes those lesions with Arteriovenous aneurysm and lymphangiomas

- Spontaneous regression is seen in some lesions at an early age (50%)
- Bleed excessively if traumatized.

Encephalotrigeminal angiomatosis

- This condition is also known as Sturge-Weber disease
- Is a congenital condition, characterized by leptomeningeal “angiomas”, nevus flammeus or port wine nevus of the face following a trigeminal distribution and cerebral calcifications
- Is uncommon
- Shows no sex predilection
- Is present at birth
- Shows ocular and neurologic symptoms in addition to aesthetically disturbing port wine nevus
- Skin, mucosa and eye lesions are clinically seen.
- Port wine nevus follows trigeminal distribution. Gingival enlargement may be seen due to increased vascular component
- X-rays show intracranial calcifications. Ocular changes include glaucoma and angioma. Hemiplegia, convulsive disorders may be present.
- With neurological features and the typical distribution of the lesion clinical diagnosis is not difficult
- Condition is benign in so far as the cutaneous or mucosal lesions are concerned
- Neurological features present complications
- Anti convulsive treatment and neurosurgical procedures are necessary.

Hereditary hemorrhagic telangiectasia

- Also called Rendu-Osler-Weber disease
- Is a congenital hereditary disease characterized by numerous telangiectatic (angiomatous) areas on skin and oral mucous membrane with hemorrhagic tendencies
- Is uncommon
- Both sexes in this condition are equally affected.
- Is congenital. Becomes conspicuous at puberty
- Epistaxis or oral bleeding may be the earliest complaint
- Skin lesions are common on face, neck and chest. Intraorally, lips, gingivae, buccal mucosa and palate are involved
- Spider like telangiectasia are angiomatous lesions which are small, slightly elevated, smooth, bright red lesions (Fig. 16.5)
- Bleeding time and clotting time is normal in this disorder



Fig. 16.5: Hereditary hemorrhagic telangiectasia. Note the raised pin-pointed red vascular lesions on the tongue

- Spontaneous bleeding may present severe complications
- Differential diagnosis includes those conditions which have a bleeding tendencies. Clinical picture mimics purpura as well
- Bleeding may be controlled by diathermy.

Lymphangioma

- Is characterized by spaces which contain lymph or serous fluid, and are lined by a single layer of endothelium
- Is not uncommon
- Shows no sex predilection
- Majority of lymphangiomas are present at birth. May become conspicuous in childhood
- Intraorally, tongue is the commonest site. Palate, gingivae and lips are also involved
- If superficial, the lesion assumes a papillary appearance. Lesions are usually of the same color as the adjacent mucosa. Deeper lesions are more diffuse and swollen. Tongue presents macroglossia and pink enlarged projections
- Differential diagnosis includes Hemangioma
- Tendency to recur after removal is seen
- Surgery is often the choice.

Cystic hygroma

Is characterized by lymphangiomatous involvement of deeper and more extensive structures of the lateral neck musculature.

- Is not uncommon
- Both sexes are equally affected in this condition
- Is seen at variable ages after birth

- Any point in the neck from base of the skull down to mediastinum is the location for this lesion
- Characterized by soft fluctuant swelling, solitary or multiple, at any point as stated above
- Aspirated fluid froths readily on agitation suggesting cystic hygroma (lymph contains a high lipid concentration)
- Differential diagnosis includes soft and fluctuant swellings of the neck
- Respiratory complications, due to airway obstruction, may be seen in this condition
- Surgery is often the choice.

Multiple neurofibromatosis

Is also called von Recklinghausen's disease of skin or Fibroma Molluscum.

- Is characterized by numerous nodular skin lesions associated with melanin pigmentations of skin
- Predominantly, the disorder is hereditary being transmitted as a dominant mendelian characteristic
- Is not uncommon
- Shows slight predilection for men
- Seen in Infancy or childhood
- Asymptomatic other than for aesthetic reasons.
- Skin—chiefly trunk, face and extremities show the lesions
- Nodular form, multiple, sessile or pedunculated nodules of variable size
- Diffuse form—diffuse lesions giving rise to a form known as “Elephantiasis Neuromatosa”
- Majority present pigmentation of skin known as “Café-au-lait” spots (coffee colored patches)

- Oral-nodular lesions are sometimes seen on buccal mucosa, palate and tongue
- “Macroglossia” may be present in diffuse involvement
- Intrabony involvement has been reported
- Generally condition does not present difficulties in diagnosis
- Malignant degeneration may occur in any nodule (15%)
- No definitive treatment because of the number of lesions is too great. Only on the face and in areas subjected to friction may be surgically removed.

Fibromatosis gingivae

Also called Elephantiasis Gingivae or Hereditary Gingival Fibromatosis.

- Is characterized by a diffuse overgrowth of the gingival tissue (Fig. 16.6), which is transmitted through a dominant autosomal gene and thus is an hereditary condition



Fig. 16.6: Fibromatosis gingivae

- Is uncommon
- Both sexes equally affected in this condition
- Seen in early childhood
- Condition is symptomless
- Gingival tissue of one or both arches is affected
- Dense, diffuse, smooth or nodular overgrowth of gingival tissue, usually appears at the time of eruption of permanent incisors
- Enlarged tissue is pale and no signs of inflammation seen
- Crowns of erupting or erupted teeth are hidden
- May result in delayed eruption
- Differential diagnosis includes Dilantin and Nifedipin and Cyclosporin associated hyperplasia and inflammatory hyperplasias of gingivae
- Tooth eruption is impeded. Aesthetically unpleasant
- Cosmetic appearance requires surgery.

Lingual tonsil

This is a reactive lymphoid aggregate and also known as Foliate Papillitis (Fig. 16.7).

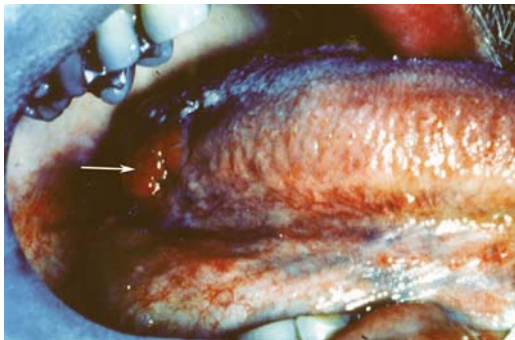


Fig. 16.7: Lingual tonsil (arrow)

- Lingual tonsil is the largest of oral lymphoid aggregates situated on the dorso-lateral aspect of tongue
- Is common
- No sex predilection is seen in this condition
- Is seen in any age. Generally in adulthood
- Condition is asymptomatic unless infected
- Posterior portion of the dorso-lateral aspect of tongue is the site of its location
- Bilateral, enlarged reddish lesion surrounded by crypt
- If unilateral, may be mistaken for carcinoma.
- Very rarely malignant Lymphomas have been reported in these lesions.

No treatment is necessary. Reassurance of the patient is essential.

B. Lesions with normal tissue in ectopic position

Fordyce's granules

Also known as Fordyce's disease (should not be considered as a disease entity.)

- Is characterized by heterotopic collections of sebaceous glands at various sites in the oral cavity.
- Very common. 80 percent of the population
- No significant difference between sexes is seen
- Become apparent at puberty (hormone dependent?)
- Asymptomatic
- Buccal mucosa, labial mucosa, tongue and gingiva are the common sites (Fig. 16.8)
- Seen as small yellow spots with a granular appearance



Fig. 16.8: Part of the left buccal mucosa showing numerous Fordyce's granules (arrows)

- Found bilaterally symmetrical. Occasionally as plaques
- Their typical distribution and clinical appearance do not present any diagnostic problem
- These are innocuous glands
- No treatment is necessary.

Lingual thyroid

Is an anomalous condition in which follicles of thyroid tissue are found in the substance of the tongue.

- Is uncommon
- Predominantly seen in females
- Becomes manifest during puberty
- Condition is asymptomatic. Often dysphagia, dysphonia, or dyspnoea may be present

- Base of the tongue is the usual site. Seen as nodular mass in or near the base of the tongue near foramen caecum. Deeply seated and is a small mass of about 2-3 cm in diameter
- If normal thyroid gland is not palpated in these patients in its normal place, then a Scintiscan with a tracer dose of radioactive iodine 1131 should be carried out to determine whether or not the 'par' lingual thyroid tissue is the only thyroid tissue present
- Should be differentiated from lingual tonsil or accessory salivary glands
- Surgical excision with proper thyroid management is essential.

Note: Thyroid nodule is derived from the thyroid anlage which failed to migrate to its predestined position.

C. Persistence of embryonic structures and/or failure of their proper development

Congenital lip and commisural pits and fistulas

Malformations of the lips often following a hereditary pattern and may be associated (often) with malformations of palate and lips.

- Are uncommon
- No data on sex predilection is available. When associated with other syndromes sex predilection may be seen
- Are congenital
- Differential diagnosis should include those conditions derived from trauma.

Double upper lip

This is a developmental anomaly resulting in folding of lip from the inner aspect (Fig. 16.9).



Fig. 16.9: Double upper lip

Rarely double lip may occur with drooping of eyelid and nontoxic enlargement of thyroid gland. This syndrome is known as Ashers Syndrome.

Double lip may be corrected by surgery for esthetic reasons.

Ankyloglossia

Ankyloglossia is also known as tongue tie (Fig. 16.10). This occurs due to a short or anteriorly attached lingual frenum.



Fig. 16.10: Ankyloglossia (tongue-tie)

The condition may be partial or complete resulting in difficulties in speech and deglutition. Surgery is performed to relieve the tongue.

Median rhomboid glossitis

Also known as central papillary atrophy this occurs anterior to foramen caecum in midline of the dorsal surface of tongue. Lesion is smooth, pink, and nodular (Fig. 16.11).

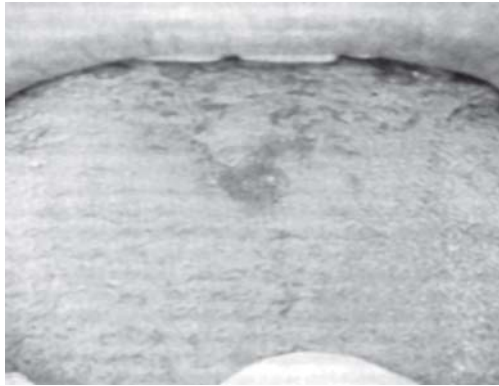


Fig. 16.11: Median rhomboid glossitis

Median rhomboid glossitis is believed to occur as a result of failure of tuberculum impar to withdraw at the time of development of the tongue but this theory is now being questioned. Current thinking is that candida organisms are thought to be the cause.

Any symptoms and treatment not necessary. Antifungal therapy is often useful if the lesion becomes symptomatic.

Migratory glossitis

This is also known as geographic tongue because of its map like appearance (Fig. 16.12).



Fig. 16.12: Migratory glossitis (geographic tongue).
Tongue also shows fissures

Condition is characterized by the presence of multiple depapillated erythematous patches which occur on the dorsal surface of the tongue. The symptomless lesions 'migrate' from one location to the other hence the term migratory has been used.

Fissured tongue

Fissures arise from the central groove on dorsal surface of tongue. Sometime these are multiple and give a scrotal appearance (Fig. 16.13).

- May occur in Melkerson—Rosenthal syndrome
- No treatment is necessary but fissures must be kept clean so as to avoid infection.



Fig. 16.13: Fissured (scrotal) tongue

Bilateral complete cleft of the primary palate

This is a cleft of the lip and alveolar ridge on both sides extending through the triangular primary palate (Fig. 16.14).



Fig. 16.14: Bilateral complete cleft of the primary palate

A congenital anomaly arising out of failure of fusion of the medial nasal prominence with the maxillary prominence on both sides. In this condition communication with the nasal cavity is established and speech, esthetics, and feeding becomes a problem. Surgery in stages is indicated.

Unilateral complete cleft of the primary palate

- This condition is characterized by cleft of the lip, alveolar ridge, and primary palate on one side resulting in communication with the nasal cavity (Fig. 16.15)
- This is a congenital anomaly due to failure of fusion of medial nasal prominence with the maxillary prominence. Speech, esthetics, feeding is a problem and teeth in the area of the cleft may be missing, e.g. lateral incisor. Surgery in stages is the main treatment.



Fig. 16.15: Unilateral complete cleft of the primary palate

Complete cleft of the soft palate

- Complete cleft of the soft palate is congenital anomaly of the soft palate due to failure of fusion of the palatine shelves during development resulting in communication with the nasal cavity (Fig. 16.16)
- Speech and deglutition are impaired as the oral contents may be regurgitated into the nasal cavity. Infection of the nasal cavity may occur. Surgery is indicated.

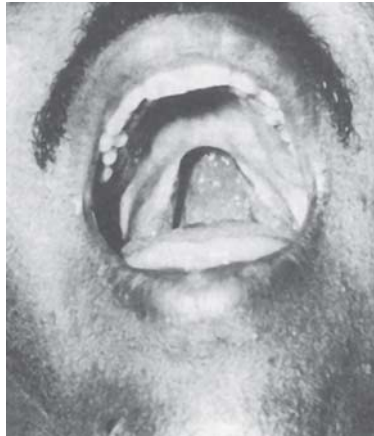


Fig. 16.16: Complete cleft of the soft palate

Complete cleft of the primary and secondary palate

This is a congenital anomaly arising out of failure of fusion of the medial nasal prominence with the maxillary prominence on both sides and the fusion of the palatine shelves (Fig. 16.17).

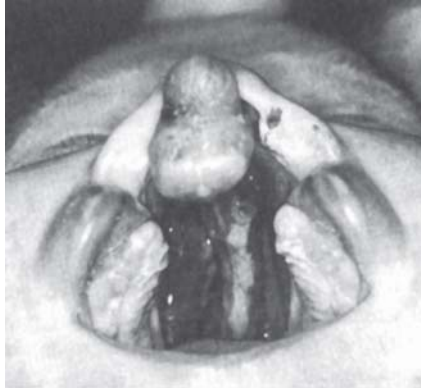


Fig. 16.17: Complete cleft of the primary and secondary palate

Communication with the nasal cavity exists and speech, esthetics, feeding are impaired. Teeth in cleft area may be missing. Surgery in stages is indicated.

Suggested reading

1. Sawyer D. In Prabhu SR, et al (Eds): Developmental Disorders of the Teeth: Oral Diseases in the Tropics. Oxford Medical Publications, Oxford 1992.

CHAPTER

17

**Radiographic
Differential
Diagnosis of
Lesions of the
Jawbones**

SR Prabhu

Learning objectives

After studying this chapter the student should be able to:

- Understand the radiological difference between radiolucent and radiopaque appearances of the jaw lesions
- Understand the basis for different types of radiolucent and radiopaque appearances that are found in the jaw lesions.
- List radiolucent lesions of the jawbones
- List radiopaque lesions of the jawbones
- List mixed radiolucent and radiopaque lesions of the jaws.

Introduction

Although advanced imaging techniques are becoming increasingly available in recent years conventional radiography will remain an integral part of dental practice. The diagnostician is therefore expected to possess adequate knowledge of interpretation of different radiographic appearances of the teeth and jawbones both in normal and pathologic conditions. This chapter is meant to introduce the student to basic aspects of radiographic differential diagnosis of the jawbone lesions. For a detailed knowledge the student is recommended to refer to standard textbooks on Maxillofacial Radiology.

Basics and pre-requisites of radiographic interpretation

Firstly, the student is expected to possess adequate knowledge of radiographic anatomy and indications and types of different views used in conventional radiography of the maxillofacial skeleton.

It is important that the radiographic evaluation is carried out systematically. The basic requirements in this regard include:

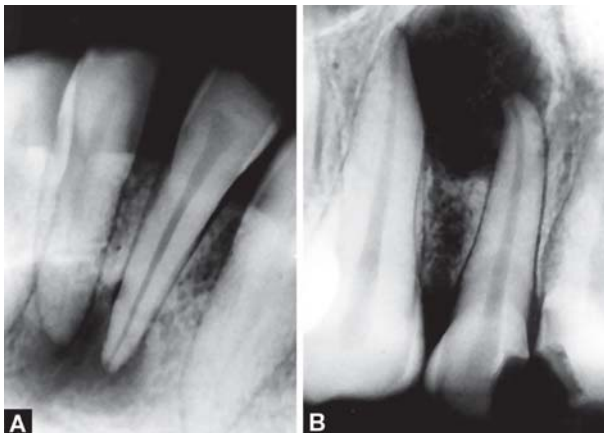
- *Identification of the patient:* Identify the patient by

checking (on the radiograph) patient's name, age, hospital number and date of exposure

- *Orientation of the radiograph:* Determine between the left and right side of the patient. Metallic markers (L and R) used for the extra oral radiographs provide this information
- Use adequate source of light and viewing conditions. (Do not read the radiographs against day light through the window or in the corridors!)
- Identify normal anatomical land marks on the radiographs
- Study the entire radiograph regardless of the striking radiographic lesion seen on the radiograph
- List all radiographic abnormal appearances seen on the radiograph. These point to the lesion(s) present.

A systematic record of the abnormal appearances should be kept. Radiographic lesions should be recorded using the following protocol:

1. *Site* (location) of the lesion (e.g. left ramus or angle of the mandible).
2. *Size* of the lesion (in millimeters or centimeters; do not use 'peanut' size).
3. *Shape* of the lesion (e.g. oval, round, pear shaped, "brandy glass" shaped, saucer shaped, etc.)
4. *Symmetry:* (If the lesion is bilateral compare both sides for symmetry)
5. *Nature and content of the radiographic appearance of the lesions*
 - *Radiolucency (Figs 17.1A-17.12)*
Lucent lesions present as single (unilocular) or multiple (multilocular) cavities. They appear



Figs 17.1A and B: **A.** Apical radiolucency resulting from chronic suppurative periodontitis. **B.** Periapical radiolucency due to pulpal pathology in lateral incisor (maybe a granuloma or radicular cyst)



Fig. 17.2: Well circumscribed radiolucency in an edentulous area of the mandible (residual cyst)

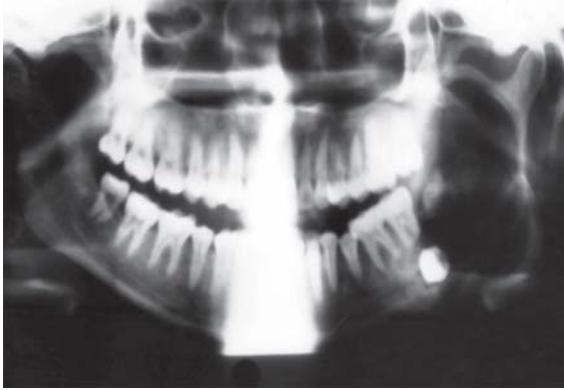


Fig. 17.3: Extensive radiolucency enclosing the crown of an unerupted lower left third molar involving left mandibular ramus (Dentigerous Cyst)



Fig 17.4: Radiolucent area situated over the crown of molar tooth in left mandible (histologically confirmed as Calcifying Epithelial Odontogenic Tumor)

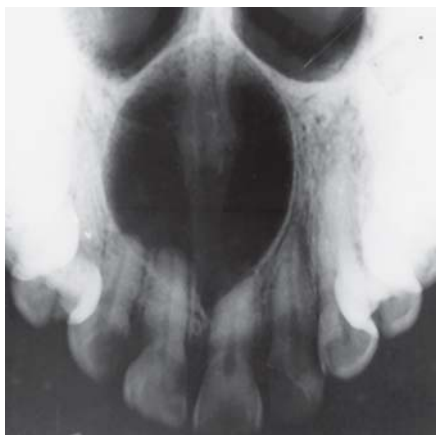
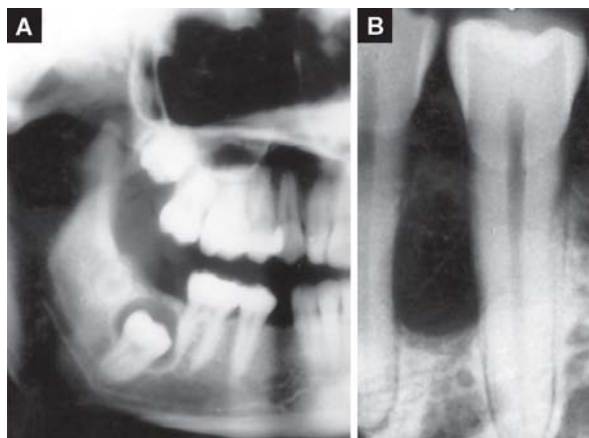


Fig. 17.5: Symmetrical radiolucency in the midline of the anterior maxilla (Nasopalatine Duct Cyst)



Figs 17.6A and B: **A.** Radiolucency surrounding crown of right mandibular second permanent molar (Dentigerous Cyst). **B.** Radiolucency abutting the root surface of teeth (Lateral Periodontal Cyst)

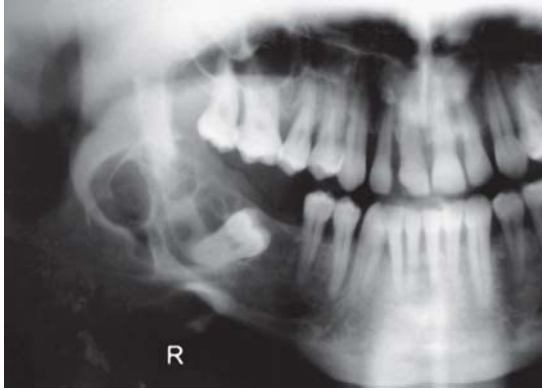


Fig. 17.7: Multilocular radiolucency at the angle of the right mandible associated with unerupted molar tooth (histologically confirmed as Ameloblastoma)



Fig. 17.8: Multilocular radiolucency of the right mandibular ramus (histologically confirmed as Odontogenic Keratocyst)



Fig. 17.9: Multilocular radiolucency of the mandible presenting 'honeycomb' appearance (histologically confirmed as Odontogenic Myxoma)

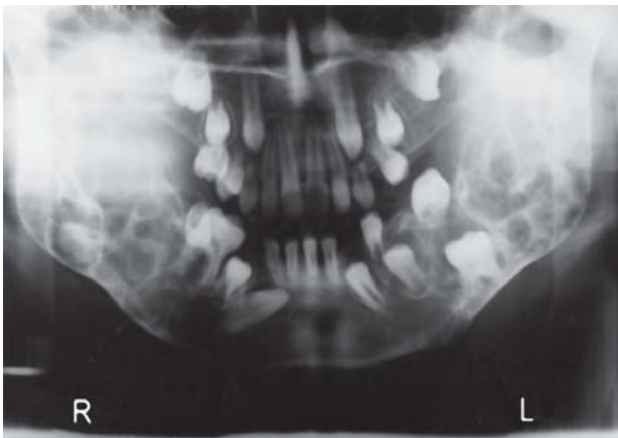


Fig. 17.10: Bilateral multilocular radiolucency presenting 'soap bubble' appearance with expansion of cortices in the body of right and left mandible (Cherubism)



Fig. 17.11: Multilocular radiolucency of the mandible in a young boy due to Burkitt's lymphoma



Fig. 17.12: Radiolucency of the left mandible showing scalloped appearance of the superior border close to teeth (Hemorrhagic Bone Cyst)

dark on the radiograph implying replacement of normal bone with non-mineralized tissue or material. Cysts and some tumors present radiolucency (see below)

- *Radiopacity (Figs 17.13-17.17A)*
Radiopaque lesions appear whiter on the radiograph implying increased density of mineralization as compared to the surrounding normal bone. Retained roots, impacted teeth, odontomas and metallic foreign bodies are some examples of radiopaque lesions (see below)
- *Mixed radiolucent and radiopaque (Fig. 17.17B)*
Lesions that present a combination of radiolucency and radiopacity suggest presence of both (greater amounts of) mineralized and non-mineralized tissues. Examples include cemento ossifying fibroma and odontomas in early phase of development (see below).

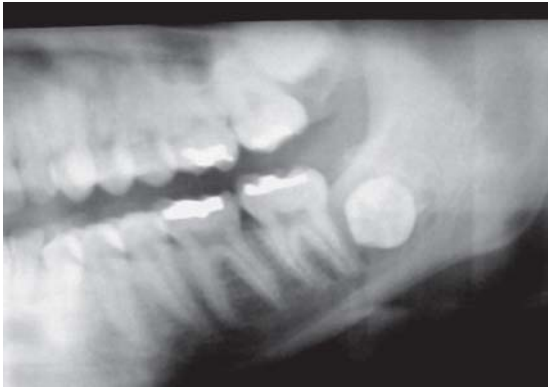
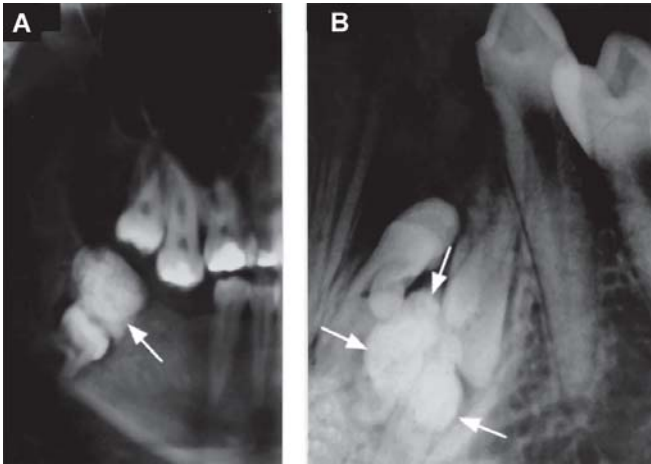


Fig. 17.13: Radiopacity in the left mandible posterior to the second molar (Bucco-lingual Impaction of Third Molar)



Fig. 17.14: Radiopacity involving the right maxillary sinus due to a malignant tumor



Figs 17.15A and B: **A.** Radiopacity [arrow] overhanging the crown of mandibular molar (Complex Odontome). **B.** Radiopaque tooth-like structures (denticles) indicating the presence of a Compound Odontome [arrows]

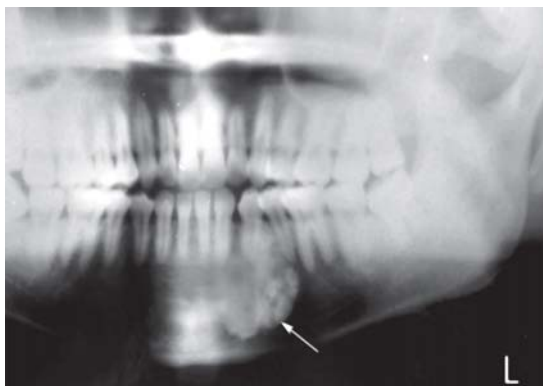
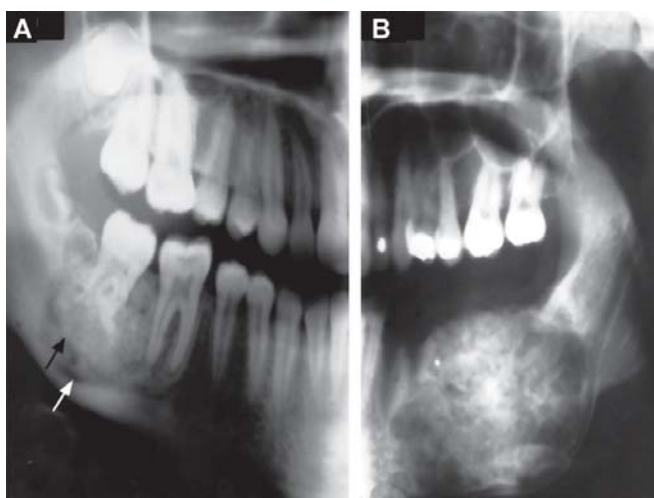
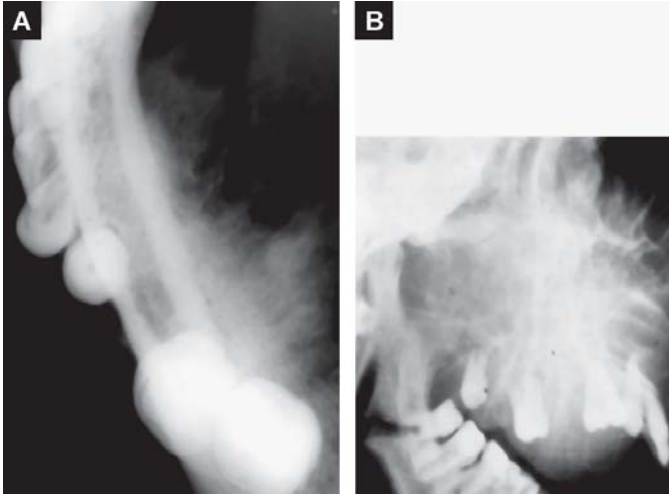


Fig. 17.16: Radiopaque (sclerotic) lesion [arrow] resulting from ossifying/cementifying fibroma



Figs 17.17A and B: **A.** Radiopaque lesion [black arrow] at the roots of the lower left second molar surrounded by a radiolucent rim [white arrow] (Cementifying Fibroma). **B.** Radiopacity mixed with radiolucency on the right mandible (Cementifying Fibroma)

- *Radiographic lesions should be further studied for:*
 1. Their border characteristics such as:
 - *Outline:* Whether well demarcated, poorly demarcated, indistinct, with a sclerotic margin, infiltrative, etc.
 - *Loculations:* Whether unilocular, multi-locular, etc.
 - *Bone rarefaction:* In rarefaction the bone is less dense as compared to normal bone, familiarize with the radiographic appearance of normal bone density first
 2. Other lesional descriptive appearances that are often used relate to trabecular patterns are:
 - *Honey-comb appearance (Fig. 17.9)*
In these situations bony septa are narrower with angulations as in odontogenic myxomas and intraosseous hemangiomas
 - *Soap bubble appearance (Fig. 17.10)*
This appearance shows septa creating varying degrees of multi-locularity, and loculi are few in number and are large and round. Seen in ameloblastomas, central giant cell granuloma and odontogenic keratocyst
 - *Sun-burst/Sun-ray appearance (Figs 17.18A and B)*
Here the trabeculations within and surrounding the lesion look like sun rays



Figs 17.18A and B: ‘Sun-ray’ appearance of chondrosarcoma of the mandible and osteosarcoma of the maxilla respectively

on the radiographs. These represent new bone that is productive or destructive but is uncoordinated from the needs of the bone as seen in Osteosarcoma.

- *Hair-on-end appearance (Fig. 17.19)*
In this appearance coarse trabeculations are seen on the skull as in hemolytic anemias, thalassemias and sickle cell anemia.
- *Moth eaten or worm eaten appearance*
Moth eaten or worm eaten appearance shows ragged border and is often seen in severe inflammations or malignancy.



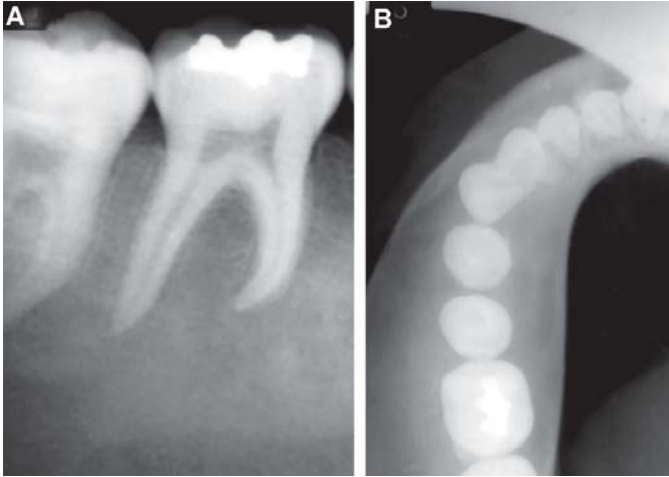
Fig. 17.19: 'Hair-on-End' appearance of the skull in thalassemia

- *Cotton wool or Cotton ball appearance*
In this appearance dense patches of sclerotic bone are formed in response to local inflammatory stimulus as in condensing osteitis. These also can be wide spread as in Paget's disease.
- *Orange peel appearance*
In this appearance dense and fine bone trabeculations are seen giving rise to an 'orange peel' appearance on intra oral radiographs. In late stages of fibrous dysplasia this appearance is seen.
- *Snow-storm appearance (Fig. 17.20)*
This appearance is a characteristic sialographic feature of the parotid gland in Sjogren's syndrome suggesting punctate sialectasis of the gland.



Fig. 17.20: 'Snow-storm' appearance of the parotid gland (Sjögren's Syndrome)

- *Ground glass or Frosted glass appearance (Figs 17.21A and B)*
Lesion with an orange peel appearance (as seen on intra oral radiograph) shows a ground glass or frosted glass appearance on the extra oral radiograph. This difference is due to the fact that resolution of extra oral radiograph is lower than that of the intra oral radiograph.
 - *Punched out appearance*
Punched out appearance is well defined with non corticated margins. Seen in histiocytoses and myeloma.
6. *Radiographic signs of the effect of lesion on other structures:* Look for root resorption, displacement of teeth, involvement of the mandibular canal, perforation of the maxillary antrum, etc.



Figs 17.21A and B: **A.** 'Ground-glass' appearance of the bone. Note absence of lamina dura in the periapical view and **B.** Bony expansion of the occlusal view (histologically confirmed as Fibrous Dysplasia)

7. Correlate radiographic appearances and findings with diagnostic information.
8. Offer a radiographic differential interpretation.
9. Integrate the differential diagnosis with other available information to form a working diagnosis and
10. Determine whether further investigations such as biopsy, blood chemistry, etc. are required. If required, order them.

It must be remembered that the radiographs are important diagnostic aids and the radiographic image interpretation is only one factor in the final diagnosis.

It must also be remembered that the radiographs are two dimensional representations of 3 dimensional structures. Often more than one radiograph with different views/angles become necessary to arrive at a working radiographic diagnosis. In some instances advanced imaging techniques need to be used for further evaluation.

Differential diagnosis of jawbone lesions

In the following paragraphs an attempt is made to briefly list some of the lesions that present above mentioned radiographic appearances. Intention is to provide a list of differential diagnosis only. A detailed discussion on the clinical entities listed is beyond the scope of this chapter. The student is recommended to refer to standard textbooks on Maxillofacial Radiology or Pathology for in depth knowledge of the pathological entities listed.

Unilocular radiolucent lesions

1. Cysts and cyst like lesions:

- Radicular (periapical, dental) cyst
- Residual Radicular cyst
- Dentigerous cyst (pericoronal radiolucency)
- Lateral Periodontal cyst
- Naso-palatine duct cyst
- Simple bone cyst (solitary bone cyst)
- Calcifying Odontogenic cyst (early).

2. Tumors:

- Calcifying Epithelial Odontogenic Tumor (early lesion)
- Adenomatoid Odontogenic tumor (early lesion)
- Unicystic ameloblastoma

- Odontogenic Keratocyst/Primordial cyst
- Primary osteolytic bone tumors (indistinct borders)
- Bone tumors metastatic to the jaws. (primary sites being, breast, kidney, lung, prostate, colon, stomach, etc) (ill-defined borders)
- Multiple myeloma
- Primary intraosseous squamous cell and mucoepidermoid carcinomas (ill-defined borders).

3. *Other pathologies:*

- Periapical granuloma
- Giant cell granuloma
- Eosinophilic granuloma (ill-defined borders)
- Fibro-cemento-osseous lesions (early lesions)
- Stafne's bone cavity
- Osteomyelitis (ill-defined borders)
- Osteoradionecrosis (ill-defined borders)

Multilocular radiolucent lesions

- Ameloblastoma
- Central giant cell granuloma
- Giant cell lesion of hyperparathyroidism
- Cherubism
- Odontogenic myxoma
- Odontogenic keratocyst
- Aneurysmal bone cyst
- Central hemangioma of the jawbones
- Vascular malformations
- Central neurofibroma.

Radiopaque lesions

- Retained roots

- Impacted tooth
- Embedded tooth
- Exostosis
- Enostosis
- Mandibular and maxillary tori
- Chronic infection leading to Sclerosing osteitis (Condensing Osteitis)
- Sequestra of osteomyelitis
- Calcifying epithelial odontogenic tumor (late stage)
- Calcifying odontogenic cyst (late stage)
- Ossifying fibroma (late stage)
- Odontomes (late stages)
- Adenomatoid odontogenic tumor (late stage)
- Gigantiform cementoma
- Cemento ossifying fibroma (late stage)
- Benign cementoblastoma (late stage)
- Periapical cemento osseous dysplasia (late stage)
- Osteopetrosis
- Osteoma
- Chondrosarcoma
- Osteosarcoma
- Metallic foreign bodies
- Ameloblastic fibro odontoma (late stage)
- Garre's osteomyelitis
- Periapical cemental dysplasia
- Hypercementosis
- Paget's disease
- Salivary gland calculi
- Antrolith
- Rhinolith
- Mineralized stylohyoid ligament as in Myositis Ossificans
- Calcified cervical lymph nodes
- Phlebolith

- Mucosal cyst in the maxillary sinus
- Objects on the face such as ear rings.

Mixed radiolucent-radiopaque lesions

- Fibrous dysplasia
- Paget's disease
- Cementoossifying fibroma
- Osteosarcoma
- Chondrosarcoma
- Compound odontoma
- Adenomatoid odontogenic tumor
- Calcifying epithelial odontogenic tumor
- Fibrocemento osseous lesions (intermediate stages).

It must be noted that the radiographic appearances often depend on the stages of development of some of the lesions listed. A lesion that is radiolucent in the early stage of development may show radiographic signs of mixed radiolucent-radiopaque or radiopaque appearance during its intermediate and late stage of development respectively.

Suggested reading

1. Goaz PW, White SC. Oral Radiology: Principles and Interpretation. Mosby, St Louis 1992.
2. Wood NK, Goaz PW. Differential Diagnosis of Oral and Maxillofacial Lesions (5th edn). Mosby, St Louis 1991.

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